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The Study and Treatment of Heart Disease at the Massachusetts General Hospital from 1821 to 1936

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IN October, 1846, ninety years ago, a young laborer named Patrick Hurley entered Ward 29 of the Massachusetts General Hospital for the relief of palpitation and breathlessness. These symptoms had developed in June and had forced him to give up working in September. For three weeks he had been under the care of a physician, who had failed to give him relief by a blister over the heart. On admission to the hospital he was found to have a rapid pulse, which was very irregular in both rhythm and force. Apparently there was a musical murmur at the apex of the heart though its description was very vague. No diagnosis was made; in retrospect now it is evident that he was suffering from rheumatic heart disease with uncontrolled arrhythmia.

An astonishing story follows: For over four months this young man was kept in the hospital with apparently no benefit whatsoever and in March, 1847, he was discharged still suffering from tumultuous rapid, irregular heart action. No chart was kept, nor was any temperature taken; the clinical thermometer and charts were later innovations. Rest, vegetable diet and the old standby of those days, bichloride of mercury, were ordered at the beginning of his stay, and throughout the months there was no change except that the mercury bichloride solution was gradually increased in dosage from ten drops to forty drops three times daily. Finally, the doctors were content when in November the patient complained of sore gums. The mercury was then suspended and a dose of castor oil was given. We can conclude at least that mercury does not help this kind of heart trouble and we can be grateful that we did not live in the days of such heroic treatment.

Little or no advance in the understanding of heart disease in this hospital is evident from the perusal of the patients' records during the first twenty-five years of the existence of the institution, which opened its doors in 1821, or in the next twenty-five years, following the introduction of ether anesthesia for surgical operations. Those fifty years were in this respect a sterile period, here, as they were all over the world, despite certain brilliant contributions of which I shall speak shortly.

In the spring of 1822, the hospital's first year, a lad of 15 years was admitted with a complaint of malaise and palpitation and a story of pains in the legs off and on for several years. His heart was enlarged and to aid in its examination the new-fangled stethoscope was used. The inexperience of the times and the difficulty of hearing much of anything through the old wooden cylinder rendered the attempt as useless as the efforts of the doctors in the professional ballad of Oliver Wendell Holmes, which I shall quote shortly. The report of the auscultation was that the heart "sound was not great", whatever that means. A blistering plaster was laid on the region of the heart, the ubiquitous bichloride of mercury was ordered daily, and a diet was prescribed which consisted of bread and milk night and morning, plain pudding for dinner and meat once in three days. Two weeks later the blister was suppurating and keeping the patient awake at night. In another two weeks when the blister had dried the terse order was given, "Let it be renewed." Six weeks after his entrance to the hospital it was noted that he was more pale than when he entered and somewhat emaciated. At least there is the virtue of frankness here. Finally, three months after admission he was discharged without comment or record of any diagnosis. Reading between the lines we may infer that here as well as elsewhere there was still medieval treatment for an unrecognized acute rheumatic infection involving the heart.

In July, 1875, a carpenter named John Reid, 52 years old, was admitted to the hospital for so-called valvular disease of the heart. The history now contains some notes about diseases in the family and a little more detail in the past history of the patient himself. His present complaint was of shortness of breath increasing over a period of six months and of swelling of his feet and ankles for five weeks past. Examination revealed great breathlessness on the slightest exertion, requiring his sleeping in a chair at night. He showed an enlarged

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heart, murmurs of aortic and mitral valvular insufficiency and swelling of his legs. A minute amount of the tincture of digitalis was ordered and increased slowly, never to an adequate amount and entirely without any obvious benefit up to the time of his death from increasing dropsy eight days after entering the hospital. Gin was given in a futile attempt to turn the tide or else to make his last days more endurable, and finally his feet were punctured as a last resort. Much serum flowed out but he went rapidly on to his death. With adequate digitalis and diuretic therapy this man would almost certainly have survived that particular spell of heart failure.

It is of course very easy in this age to criticize the treatment and diagnoses of those days in 1822, 1846 and 1875. I dare say our successors fifty to one hundred years from now will look back with equal surprise and incredulity at our gross ignorance and apparent neglect. But it is truly astonishing to realize the vast strides that have been made in our own generation in the understanding of medical science, as exemplified by the heart and its diseases. Without any doubt the advance in this field has been greater in the last fifty years than in all the centuries before. And in this advance the Massachusetts General Hospital has made its share of contributions.

It is of the greatest interest to peruse the diagnosis index of the volumes of the records of the ward patients in the first third of the hospital's existence, that is, from 1821 to 1860, and to note the medical diseases with which the staff had to cope. First place was taken by pulmonary tuberculosis with 33 solid pages of names of patients, which averaged about 35 names to a page. A close second was typhoid fever with 32 pages. Then came acute rheumatism with 21 pages, dyspepsia with 18, chronic rheumatism with 13, pneumonia 11, bronchitis 9, unspecified fever 9, diarrhea 6, syphilis 5, dysentery 4 and only scattered recognized cases of such conditions as troubles with the nervous system, the heart and the kidneys. There were no complete and satisfactory cardiac diagnoses; those that were given were anasarca, cardialgia, carditis, dyspnea, endocarditis, dilatation of the heart, hypertrophy of the heart, palpitation of the heart, disease of the heart, edema, pericarditis and one case of angina pectoris. Between 1860 and 1870 3 more cases of angina pectoris were added, one case of aortic regurgitation, and one of rupture of the aorta.

In contrast the cardiac diagnoses of the one year 1934 led all the rest with 1,261 cases, founded so far as possible on the tripod of cause, structural change, and functional condition. Tuberculosis of the respiratory system was diagnosed in 212 cases that same year, typhoid fever in only 8, acute rheumatic fever in 51 and chronic rheumatism in 246.

The great increase in the importance of the heart problem at the Massachusetts General Hospital, both absolutely and relatively, in the last century is well illustrated by these figures. Here is certainly one reason why the great advance in our knowledge of heart disease has occurred; we have been forced by its very magnitude to put more workers on the job, to spend more and more of their time.

Before leaving the dark period of ignorance about the heart of one hundred years ago I would pay tribute to several brilliant contributions made by observant physicians prior to that day, contributions that flashed across the sky, only to be ignored as a rule, or quickly forgotten, or but little applied to the practice of medicine.

Benivieni and a few of his contemporaries in Italy about 1500 had shown for the first time that heart disease was possible without causing instant death. Realdus Columbus in the middle of the sixteenth century taught the circulation of blood through the lungs and in 1628 Harvey completed the work of the Italian physiologists with his masterly treatise. In 1733 an English parson named Hales measured blood pressure for the first time, but no further attention was paid to this for a hundred years and the idea was not utilized in medical practice for nearly two hundred years. In 1715 Vieussens described in detail the coronary arteries, in 1768 Heberden introduced the term angina pectoris, and at the end of the eighteenth century Jenner of smallpox fame put together these two observations, namely, angina pectoris and the coronary arteries. In 1728 Lancisi of Rome introduced the conception of heart failure and in 1832 Hope completed its fundamental principles. In 1749 Senac in Paris advised the use of quinine for rebellious palpitation, a method of treatment that was rediscovered by a patient of Professor Wenckebach in 1914. In 1775 William Withering's attention was called to the value of the foxglove in dropsy and he published a treatise of the greatest practical value in 1785, and yet one hundred years later his advice was not being followed, even here at the Massachusetts General Hospital. In 1761 Auenbrugger of Vienna introduced percussion as a method of medical examination, but for over forty years this was ignored until Corvisart, physician to Napoleon Buonaparte, advised its use in 1808, one year before Auenbrugger died. In 1819 Laennec introduced the stethoscope, but for fifty years or more this instrument was as much a hindrance as a help. The common disregard for the value of auscultation in the study of heart disease in the middle of the last century is entertainingly expressed in

a ballad by Oliver Wendell Holmes, who was Physician at the Massachusetts General Hospital from 1846 to 1849.

THE STETHOSCOPE SONG

A Professional Ballad

There was a young man in Boston town,
He bought him a stethoscope nice and new,
All mounted and finished and polished down,
With an ivory cap and a stopper too.

It happened a spider within did crawl,
And spun him a web of ample size,
Wherein there chanced one day to fall
A couple of very imprudent flies.

The first was a bottle-fly, big and blue,
The second was smaller, and thin and long;
So there was a concert between the two,
Like an octave flute and a tavern gong.

Now being from Paris but recently,
This fine young man would show his skill;
And so they gave him, his hand to try,
A hospital patient extremely ill.

Some said that his *liver* was short of *bile*,
And some that his *heart* was over size,
While some kept arguing all the while,
He was crammed with *tubercles* up to his eyes.

This fine young man then up stepped he,
And all the doctors made a pause;
Said he,—The man must die, you see,
By the fifty-seventh of Louis's laws.

But since the case is a desperate one,
To explore his chest it may be well;
For if he should die and it were not done,
You know the *autopsy* would not tell.

Then out his stethoscope he took,
And on it he placed his curious ear;
Mon Dieu! said he, with a knowing look,
Why here is a sound that's mighty queer.

The *bourdonnement* is very clear,—
Amphoric buzzing, as I'm alive!
Five doctors took their turn to hear;
Amphoric buzzing, said all the five.

There's *empyema* beyond a doubt;
We'll plunge a trocar in his side.—
The diagnosis was made out,
They tapped the patient; so he died.

Now such as hate new-fashioned toys
Began to look extremely glum;
They said that *rattles* were made for boys,
And vowed that his *buzzing* was all a hum.

There was an old lady had long been sick,
And what was the matter none did know:
Her pulse was slow, though her tongue was quick;
To her this knowing youth must go.

So there the nice old lady sat,
With phials and boxes all in a row;
She asked the young doctor what he was at,
To thump her and tumble her ruffles so.

Now, when the stethoscope came out,
The flies began to buzz and whiz;—
O ho! the matter is clear, no doubt;
An *aneurism* there plainly is.

The *bruit de rape* and the *bruit de scie*
And the *bruit de diable* are all combined;
How happy Bouillaud would be,
If he a case like this could find!

Now, when the neighboring doctors found
A case so rare had been descried,
They every day her ribs did pound
In squads of twenty; so she died.

Then six young damsels, slight and frail,
Received this kind young doctor's cares;
They all were getting slim and pale,
And short of breath on mounting stairs.

They all made rhymes with "sighs" and "skies",
And loathed their puddings and buttered rolls,
And dieted, much to their friends' surprise,
On pickles and pencils and chalk and coals.

So fast their little hearts did bound,
The frightened insects buzzed the more;
So over all their chests he found
The *râle sifflant*, and the *râle sonore*.

He shook his head;—there's grave disease,—
I greatly fear you all must die;
A slight post-mortem, if you please,
Surviving friends would gratify.

The six young damsels wept aloud,
Which so prevailed on six young men,
That each his honest love avowed,
Whereat they all got well again.

This poor young man was all aghast;
The price of stethoscopes came down;
And so he was reduced at last
To practise in a country town.

The doctors being very sore,
A stethoscope they did devise,
That had a rammer to clear the bore,
With a knob at the end to kill the flies.

Now use your ears, all you that can,
But don't forget to mind your eyes,
Or you may be cheated, like this young man,
By a couple of silly, abnormal flies.

And now we come to the days of forty years ago. Great changes began at the end of the nineteenth century. The intensive study of medical problems by capable men was beginning; no longer did all physicians do general practice—obstetrics, surgery and medicine. There was the beginning of widespread concentration in these fields, that is, the real beginning of specialization. Outstanding internists, such as Calvin Ellis, Frederick Shattuck, Reginald Fitz, James Minot, James Jackson, Herman Vickery and Richard Cabot, began to appear on the staff of the Massachusetts General Hospital and others, including Henry Jackson, George Sears, W. S. Thayer and Joseph Capps, graduated here as house officers to go elsewhere to make for themselves outstanding names as authorities in the study and treatment of cardiovascular disease.

Thus, when Andrew McGinn, a laborer 60 years old, was admitted to the hospital for study and treatment on February 13, 1900, a great change in the understanding of heart dis-

ease was in progress. In his case, family history, habits and present illness were concisely recorded. McGinn had been well until a month earlier, when shortness of breath and palpitation developed on effort. A rapid increase of these symptoms was followed by swelling of the legs during the week before admission. Examination revealed a well-developed and well-nourished man with blue lips, prominent neck veins, visible arterial pulse in the neck, enlargement of the heart accurately defined, absolute cardiac arrhythmia, blowing systolic murmur at the apex of the heart, accentuation of the pulmonary second sound, rapid pulse of high tension, moist rales at the lung bases and edema of the legs extending into the thighs. The temperature was now routinely recorded and also the respiratory rate, the blood examination and urine analysis.

Treatment included absolute rest, tincture of digitalis in adequate amounts and a diuretic drug, theobromine sodium-salicylate.

Two days after admission there was a note of great improvement. Both the breathlessness and the dropsy had almost disappeared. He had passed 173 ounces of urine in the previous twenty-four hours, and an order was written to measure the day and night amounts.

On February 20 he had improved so much that the foxglove was omitted, on the twenty-first day it was noted that his pulse was much slower and on the twenty-sixth he was up and about, completely relieved. He was discharged three days later with a diagnosis of general arteriosclerosis, interstitial nephritis (or Bright's disease) and cardiac hypertrophy with relative mitral insufficiency.

If this man were to enter the hospital today, we could hardly have treated him better. The one important addition to his treatment would be the continuation of a daily ration of foxglove permanently, an important omission until recent years in the practice of most physicians except a few very wise men, who, like Henry Jackson, realized its value even in the old days. As for routine study we would now gain some help from three sources not clinically available in 1900—blood pressure estimation, x-ray study and electrocardiography. We would recognize now that the fundamental cause of McGinn's heart trouble was high blood pressure and not kidney disease, that the uncontrolled cardiac arrhythmia, which we call auricular fibrillation, had precipitated his symptoms and that the mitral valve insufficiency was the result of his heart failure, points of fundamental importance that have become common knowledge in the years since 1900. But we would still be ignorant of the most important fact of all, namely, the cause of the high blood pressure.

I am sure that the physicians of the last generation and of our own are not more brilliant or industrious than were those of Ether Day, 1846, or before, and yet the contrast in diagnosis and treatment of patients with heart disease then and now is startling and almost incredible. What is the answer? It must be already obvious, but as I go on it will be increasingly so; I would make it the chief burden of my song.

Mention has already been made of the great clinicians of the end of the nineteenth century and of the beginning of the present century, who by their experience and contributions put the Massachusetts General Hospital in the very van of our increasing knowledge about heart disease. I would speak further of a few of these men and of their successors.

It was my good fortune while in the medical school to attend the clinical lectures on heart disease given by Dr. Frederick Shattuck. Through his rare ability, rich experience and special interest in the subject he passed on to his associates and to us sound judgment of the foremost knowledge of the day. The influence of his teaching was deep and far flung, much greater than that of anything he ever put into writing. He excelled both in diagnosis and in treatment.

But the greatest contributor in the field of heart disease in the history of the Massachusetts General Hospital, as well as in Boston, has been Richard Cabot, who is still working for the cause of the hospital and for the health of the minds, of the bodies and of the souls of this community. It is with especial pleasure, gratitude and affection that I tender this tribute to him today. He was my medical chief at home and during the World War abroad, where I learned to know him best and to appreciate at their true worth his mind and heart, which I fear have sometimes been misunderstood. It was he who worked intensively to improve our acumen in the diagnosis of heart disease, which has been one of his particular interests, witness of which is his volume, entitled, "Facts on the Heart." It was he who constantly insisted that we keep our feet on the ground in the days when our enthusiasm in the study of functional disorders, important though it was, tended to distract our attention from the study of structural pathology. His clinicopathological conferences stand as unique contributions; they have never been equalled. It was he who made the most important American contribution to cardiology, one of the greatest landmarks of all time in the history of the development of our knowledge of cardiovascular disease. Already an experienced medical leader of international reputation and so much a figure then, in 1914, that in Europe I was asked if he could still be alive, he published a paper in the *Journal of the American Medical Association*, enti-

tled "The Four Common Types of Heart Disease." This short and simple paper not only revolutionized cardiac diagnosis by introducing its most important element, namely, cause or etiology, but it forced to the attention of the medical world the absolute necessity of paying more attention to the prevention of heart disease than to either its diagnosis or its treatment. While Sir James Mackenzie initiated important progress by stirring up interest in the functional disorders of the circulation, Richard Cabot rendered much more vital service in his analysis of the fundamental factors behind heart disease. It takes a generation nearly its lifetime to appreciate some of the things its living members are doing and have done; in accord with this we are only now realizing the importance of Richard Cabot's contributions of twenty to twenty-five years ago. It is due time that we pause a moment and belatedly acknowledge our indebtedness to one who has given so much to medicine and particularly to those of us who are working in one of his chosen fields, the study of heart disease.

Furthermore, it was Cabot who instituted hospital social service in this country thirty-one years ago in his effort to help, at first, this hospital's patients, and then other patients all over the world. This service has grown so fast and so large that we can hardly follow its ramifications, but to one of these I shall refer again as it deals with the home care of children with heart disease. Finally, and, I believe, most important of all, it was and is Cabot who rightly insists that more vital than the medical care, more vital than the social service that we render our patients, is the nurture of the spiritual side of those that have entrusted themselves to us. The wise physician does this important service in his private practice almost unconsciously and thereby has a hold in his community, possible for no other member of that community except it be a rare devoted minister of the gospel. It was in large part Richard Cabot's teaching that I followed when I wrote on the "Heart of a Child" in 1933 as follows: "What is the use of all this study and work if one simply spreads the fear of heart disease and if the children whose hearts are affected grow up to be hopeless and helpless under sheltering care? It would be better if they would not survive if that is all that can be done for them. Even education consisting in training the mind alone in school curriculums, helpful and important as it is, will not suffice. Far more must be done. Things that are often neglected, especially in so-called modern times, must be given careful attention. *One must revive, nourish, and protect the spiritual heart of the child.*"

With this pioneer work of Richard Cabot's as a background may I now proceed to give you a glimpse of the experience in the further development of our knowledge of heart disease that has been the fortunate lot of those of us that have devoted our time to this field at the Massachusetts General Hospital during the past twenty years. In the course of my recital I must perforce give you some personal adventures, but these are presented in entire modesty and at the end in a word I will show why they cannot be presented otherwise.

As a fourth year medical student, I had the privilege of having some optional instruction from Joseph Pratt who was full of the enthusiasm in the newer studies of the functional disorders of the circulation handed on to him by James Mackenzie, whom he had helped to discover in the mining town of Burnley in the north of England. After my graduation from school and hospital and study abroad under this same master, Mackenzie, and his pupil, Thomas Lewis, I returned in 1914 to assist Dr. Pratt and Dr. Eustis in the newly organized adult and children's heart clinics in the Outpatient Department. Here we puzzled over obscure heart cases and here we learned that it was often better to spend a long time on one case than to feel obliged to abandon patient and solution of his problem just because the usual scheduled hour or half hour was up. I do not want to advise the upsetting of necessary schedules and programs or selfish disregard for the time of others, but I do want to say that on several occasions in the past fifteen years punctual adherence to the usual daily routine of work and play of the so-called efficient American life would have ruined our pursuit of some clinical problem that has made us all better doctors or would have left unhelped some patient who sorely needed our aid, but who required more than the usual length of time for this accomplishment.

While I was medical interne in the years 1912 and 1913 under the stimulating tutelage of Drs. Richard Cabot, William H. Smith ("Big Bill" as we called him) and Roger I. Lee, two of the new methods of study of cardiac cases were becoming firmly established. It was now for the first time the custom to measure the blood pressure in all patients and to obtain x-ray pictures of the heart whenever there was uncertainty about heart size or diagnosis. We still talked about Bright's disease to the exclusion of hyperpiesia, however; we called coronary thrombosis, angina pectoris or acute indigestion, and we had not yet learned the significance of su a condition as auricular fibrillation.

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In 1913 two events occurred, which inaugurated the new cardiac program that has followed. Mr. Skinner, a patient of Dr. William H. Smith's, through him offered to aid the progress of medical science at the Massachusetts General Hospital, and it was decided to install

in the hospital one of the new string galvanometers called electrocardiographs. This instrument, a delicate apparatus, which registers the electrical activity of the heart muscle, was in those days viewed with suspicion by most of the doctors that had ever heard of it. It was regarded as little better than the clumsy old mechanical pulse tracing machine called the sphygmograph, which by the way was asked for in 1882 by Dr. Samuel Abbott whose request was referred to the Chairman of the Trustees and which apparently was not heard of again until Dr. Joseph Pratt years later, about 1910, brought in his own Mackenzie ink polygraph to use in the Outpatient Department.

The other event was the coincidence of the date of my own graduation as interne and the date of the despatch by the new chief of the Medical Service, Dr. David Edsall, of some young doctor to London to study hearts and electrocardiography for a year and to bring back from Cambridge, England, an Einthoven string galvanometer. This instrument we installed at the hospital in the fall of 1914, in the only empty space, namely the basement of the skin ward. We built ourselves modest quarters adjoining the bathroom used by luetic patients, and here our heart work had its headquarters during the next few years until the demands of increased space carried us upstairs to the old apothecary shop on the ground floor of the Bulfinch Building under the stone steps just to the left of the central entrance door. One other move was made in 1925 to still more ample quarters, where we are now, in the new medical ell at the east end of the Bulfinch Building. To procure these ample quarters we were obliged to return again to the basement. At the present time we are again crowded and seeking more room. Interestingly enough the only available space is, as it was in 1914, in the basement of the skin ward; there we shall build ourselves an extra room or two, temporary we hope, to supplement our present quarters, until such time as the projected new building is completed.

The old electrocardiograph did yeoman's service indeed. In seventeen years it wrote 21,160 electrocardiograms and helped to demonstrate that it and all its brothers, sisters and descendants were not useless toys, but were vital additions to clinical medicine in the proper understanding of heart disease. In 1931 it was relegated to the attic and we installed a new two-string galvanometer, which has written 17,118 more electrocardiograms in the past five years. This instrument and a vertical orthodiagraphic fluoroscope for x-ray study of the heart were made available through the generosity of George Steedman, who donated a fund for cardiac research. This fund served us well for six years, but now it has finished its fruitful harvest of clinical investigations and our research balance is but a few dollars. *Verbum benevolentibus!*

I shall not weary you with a detailed recital of our studies in heart disease during the last fifteen or twenty years, but I shall take the time to acknowledge my indebtedness to the cardiac research fellows and to others who have carried on most of the work. There was Sidney Burwell, my first coworker in 1920, who is now dean of the Harvard Medical School and still not too busy to continue his interest in the circulation. Then came Harold Marvin, now at Yale and secretary of the American Heart Association. Others that followed were Edwin Wood, now Professor of Medicine at the University of Virginia, Louis Viko of Salt Lake City, Harold Segall of Montreal, Howard Sprague, now my associate, Duckett Jones of the House of the Good Samaritan in Boston and also with us, Seeley Mudd of Pasadena, California, Robert Palmer and Edward Bland, both at work here at the hospital, Paul Camp of Richmond, Oscar Brenner of Birmingham, England, Sylvester McGinn with us in Boston, Edward Orgain of Duke University, Ashton Graybiel, Earle Glendy, William Paul Thompson and Wilfrid Comeau, all of our heart group here, and Jorge Salcedo of Bogota, Colombia. We have also been blessed by other stimulating coworkers, who were not cardiac research fellows as such. These have included, among others, Edward Richardson, Arlie Bock, Joseph Aub, E. D. Churchill, J. C. White, James Faulkner, William Kerr, Merrill Myers, Hugo Roesler, George Holmes, Aubrey Hampton, Tracy Mallory, and Benjamin Castleman. It is a privilege to certify to the constant help and stimulation of our association with all the other groups and departments in the hospital and to our own devoted secretaries and technicians. Finally, we owe a great debt of gratitude to the group of women that has carried on vital, pioneer social service in connection with our children's heart clinics, namely, Misses Ida Cannon, Edith Terry, Catharine Breed and Lorena Love, and the important Committee for the Home Care of Children with Heart Disease, which was established as a very vital new venture by Mrs. Fritz Talbot in 1914.

Our work has included the routine study and treatment of cardiac patients in outpatient heart clinics for adults, for children and for adolescents, in hospital wards, in the Baker Memorial Hospital, in the Phillips House, in private homes and in our private office. One of our most interesting and useful functions has been in the teaching of graduates of medical schools that have progressed to the point of wishing further instruction in this special field.

In our investigations we have paid particular attention to the clinical significance of certain

symptoms and signs, to enlargement of the right ventricle (particularly the acute cor pulmonale), to heart failure, especially when it primarily involves the left ventricle, and to congestion of other origin, in particular that due to chronic constrictive pericarditis. The diagnostic help afforded by electrocardiograph and x-ray and by other methods of examination we have studied in detail. But *the kinds and causes of heart trouble have been our prime interest*, following the lead of Richard Cabot, for here we are aiming at our real goal, *the prevention of heart disease*, which is still beyond our reach. Congenital, rheumatic, bacterial, hypertensive, coronary disease and nervous factors are the important ones. We have helped to attack these by trying to learn more about them; in each type of heart disease different colleagues of our group have taken particular interest and are going on with a more concentrated study. It is impossible for one man now to cover adequately the whole field, and yet only fifteen years ago, when I was starting my own specialized work on heart disease, I was warned that my professional interest would become too narrow, that the field was too small. But heart disease is now the leading cause of disability and death.

Finally, our studies have included the treatment of heart disease by drugs, such as digitalis and its allies and quinidine, by physical methods, as in the use of special beds and Southey's tubes, and particularly by the development of a good mental attitude in the patient through the efforts of doctors and of social workers. Optimism and the improvement of the home care of heart patients have been keynotes.

The bulk of what has been accomplished here in heart study in the past fifteen years would have been impossible without the wholehearted cooperation of the administrative and medical staffs, in particular Drs. Washburn, Bigelow, Faxon, Edsall and Means, who have helped us tremendously *by letting us alone* to pursue our concentrated specialized work and by the frequent assignment of Dalton Scholarships to the research fellows. We have been spared the routine chores that not so long ago would have been deemed necessary for our own welfare. I herewith express my own great satisfaction at this course, which I am certain has been a wise one, and I hope it may be allowed to continue to operate in other fields besides our own. Hospital, community and medical science are all certain to benefit greatly thereby.

And now, before closing, I return to the chief motive and inspiration behind the advance of the heart work at the Massachusetts General Hospital since 1895; the first half of this period of forty years I had no hand in, but tradition had begun and it was easy to carry on. The keynotes have not been love of medicine, love of mankind and hard work, though we do ourselves confess them; essential though these things are they were a part of the life of the doctors here one hundred years ago when so little was known about heart disease. Nor has the motive been the utilization of brilliant minds, though I do agree that we have gained much in that respect from Frederick Shattuck and Richard Cabot. The answer is simple and obvious. *More and more time has been devoted to the study of heart disease by more and more persons.* When that happens we are bound to make rapid progress in an undeveloped field. We would be stupid indeed if we did not. Such is the state of affairs in every field of human activity or thought. Realizing this simple truth twenty years ago we said to ourselves that in the course of time we could place the heart work here near the apex of medical advance, if we hammered away. It has simply taken everlasting persistence. The war years interrupted the pursuit of this ideal, as war, a real relic of the dark ages, always obstructs true progress and happiness everywhere.

After the war, in 1920, we started afresh, reiterating our belief in the fundamental principle of concentrating for years on the clinical study of the circulation, a thing that even at that late date was not being done widely in internal medicine. Fortunately some parts of the body had already escaped from the domination of the omniscient professor of the Middle Ages to the great advantage of medical science and particularly of the sick man himself. The teeth, the nose and throat, the eye, the brain and nerves, the bladder, injuries, pregnancy and childbirth, all had emerged long before the heart was considered important enough to be studied in concentrated detail itself. In those early days, only sixteen years ago, it was often suggested that the field of cardiology and its name were beneath the dignity of a young physician trained in internal medicine, but the handwriting was already on the wall for those who would stop and read. I even suspect that the general consulting internist will some day cease to be; he will have served his function, a very important one in his stage of the history of medical progress, but a function that now can best be shared by family physicians and specialists, both in hospital and in private practice. Even the correlation of research is no longer a major role of the general internists; the special workers themselves are best able by their experience and intelligence to observe where their fields meet and interlace, and they have upon to cooperate in their studies. We found this to be so in our own work, joining directly with family physician, roentgenologist, surgeon, pathologist, social service worker, physiologist and neurologist in the pursuit of our

studies. Hospital and medical school services must continue to be administered by the ablest executives and teachers, as they have in the past, but always time and space and money should be spared here at the Massachusetts General Hospital for the active work of clinical investigators who are practicing medicine, men and women who are not only at work in some concentrated field at the beginning of their career when their experience is limited and not very valuable, as has been largely the custom, but also those who continue for years in their chosen field with the increasing value of their ripening experience, which rolls up like a ball of snow. Such workers are the natural selection for the teaching of progressive medical graduates, a function that is certain to assume in time its proper place as a vitally important part of medical education; the undergraduate instruction has become highly developed while the equally important graduate instruction has been allowed to grow like Topsy.

There has been a fashion in our day to overemphasize superficial and evanescent clinical research, encouraged by the flitting about from subject to subject, from city to city and from clinic to clinic after the period of the young physician's early training has passed. This course does not produce the sound results in medical knowledge and advance that concentration in one subject and in one clinic is bound to produce when one is surrounded by able workers in other fields of medical science and now and then travels about to learn what is going on elsewhere. The only real difficulty that we have encountered in our experience here has been the constant temptation to leave our work partly done and to migrate to other fields of medical endeavor, in teaching, administration or research, here or in other places, but the motive handed on, often unconsciously, by Richard Cabot, Thomas Lewis and other leaders has held us to our post. It is now becoming increasingly evident that this has been wise. In hospital clinics and in private practice there are now many patients that we, ourselves, have followed carefully for ten to fifteen years and more, and daily they are revealing to us new facts or problems and giving us the answers to questions that could not have been solved without our own personal prolonged study of cooperative and intelligent persons.

I emphasize herewith the great value of contact with private patients in medical practice and investigation, and congratulate the hospital for the part, past, present and future, played by the Phillips House and especially by the Baker Memorial. It has been my experience at both in teaching and in investigation private patients are essential for the best results. It is rather obvious that this should be so. In the first place, the higher average intelligence of the private patient permits the drawing of more accurate clinical pictures of disease; secondly, his cooperation is more readily secured for the same reason and because of the close personal relation between doctor and patient; thirdly, he is more easily followed up over long intervals of time; and finally, study after death is more often possible, with all the wealth of new information that is available thereby.

Another fact I wish to take a moment to emphasize is that we have made more progress by the simple but concentrated study of quite ordinary symptoms and signs than by the use of the newer instruments, as important as they are. It is worthy of comment that the greatest advance of all in our study of cardiovascular disease has been in history taking and physical examination and in simple observations of other nature, all possible hundreds of years ago, but neglected because no one took the simple step of devoting time intensively to them.

I would refer also to the advances made by a study of errors, both in diagnosis and in treatment. To the beginners in medicine especially I would urge the cultivation of this field; our own richest finds have come from it. Do not scorn or hide your errors! When anything unexpected or exceptional confronts you, jot it down on your list of problems to take up either at once or when you have time; that list will grow steadily and you will have some of it to hand on to your successor.

My closing word is the most important of all. It is our aim to make this hospital eventually unnecessary and to close its doors if that happy time ever should arrive. Already the hospital is unnecessary for much that it had to do a scant twenty years ago, as in the care of patients with typhoid fever and infantile dysentery. In order to effect this aim we must do more than to perfect an ideal routine for the diagnosis and treatment of disease. We must set aside ample leisure, space and money for the intensive study of the diseases of all parts of the body in the advance to our ultimate goal, the prevention of disease. Oliver Wendell Holmes wrote in 1860:

"What makes the Healing Art divine?
The bitter drug we buy and sell,
The brands that scorch, the blades that shine,
The scars we leave, the 'cures' we tell?

"Are these thy glories, holiest Art,—
The trophies that adorn thee best,—
Or but thy triumph's meanest part,—
Where mortal weakness stands confessed?

. . . . . .

"And lo! the starry folds reveal
The blazoned truth we hold so dear:
To guard is better than to heal,—
The shield is nobler than the spear!"

EVALUATION OF THE SIGNS OF ACTIVE RHEUMATIC FEVER: WITH ESPECIAL REFERENCE TO THE ERYTHROCYTE SEDIMENTATION RATE AND LEUKOCYTE COUNT*

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THE common findings during acute rheumatic fever are tender and painful joints, with or without associated swelling and redness, fever, subcutaneous nodules, precordial pain, rash (typically of the erythema marginatum type), chorea, epistaxis, and evidence of cardiac involvement. Furthermore, these clinical manifestations are usually accompanied by an elevated leukocyte count and rapid erythrocyte sedimentation rate, and sometimes a prolongation of auriculoventricular conduction time, as detected by the P-R interval of the electrocardiogram.

With recovery of the patient these clinical and laboratory signs eventually subside except for the evidences of permanent cardiac involvement. As a rule, the regression of these signs is so gradual that the disease passes from an acute to a chronic stage. In this respect rheumatic fever is more comparable to tuberculosis than to the acute infectious diseases. As long as any of these clinical and laboratory signs persist, active rheumatic fever must be considered as being present, though possibly in a low-grade form.

Since there is no specific test for rheumatic fever, it is only by the recognition of persistent and often easily overlooked manifestations that the presence of the active disease is made known. Exacerbations are frequently noted in those individuals who have been allowed physical activity soon after the acute stage of their illness. In addition, the development of heart disease during the months just subsequent to clinical symptoms of rheumatic fever is also common. It is, therefore, important to determine the duration of the activity of the rheumatic fever process.

The purpose of this report is to evaluate the various signs of active rheumatic fever, especially those by which the low-grade stage of the infection may be recognized. This involves particularly an investigation of the relative merits of laboratory tests.

MATERIAL STUDIED

The present report is based on a study of 178 patients with active rheumatic fever observed at the House of the Good Samaritan during the periods, January, 1932 to May, 1933, and September, 1934 to December, 1935. The ages varied from 3 to 27 years, and averaged 10 years.

In 163 of these cases an analysis has been made of the routine laboratory tests and a comparison made of the leukocyte count and erythrocyte sedimentation rate in respect to their value as tests for active rheumatic fever. The clinical manifestations have been analyzed as well in 73 of the patients with clinical evidence of rheumatic fever, and in these the relation of laboratory tests to the clinical signs has been determined. The frequency with which the laboratory tests were performed has varied with the individual case from once a month to twice a week.

LABORATORY TESTS FOR INFECTION

The value of the leukocyte count as a test for active rheumatic fever has been emphasized by Swift, Miller, and Boots,[1] who pointed out that an elevated count frequently persisted after all manifestations of rheumatic fever had disappeared. The erythrocyte sedimentation rate has become established as a test for infection, chiefly through the work of Fahraeus.[2] More recently this test has been applied to the study of rheumatic fever and most investigators[3] who have worked with it have acclaimed it superior to all other tests, including the leukocyte count. The popularity of the erythrocyte sedimentation rate has reached so great a magnitude that today there is a dangerous tendency to depend wholly upon it.

Before proceeding with our findings in regard to this, it is necessary to consider certain facts about the tests themselves.

THE LEUKOCYTE COUNT—Garrey and Bryan[4] recently have written an extensive review of the literature concerning the leukocyte count and its variations. They conclude that, as the leukocyte count increases above 10,000, the chances of its occurrence in a normal individual decrease rapidly. We have, therefore, accepted the arbitrary level of 10,000 as a fair upper limit of normal for the leukocyte count in the present analysis. It has been our experience that rheumatic fever subjects followed for long periods after active rheumatic fever eventually have a leukocyte count of under 10,000.

The leukocyte count is easily and quickly determined, but its accuracy is not great. This has been emphasized particularly in recent years by Brandt,[5] Ponder, Saslow, and Schr[illegible]

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Yates and Batt.[7] As a check on our accuracy duplicate determinations were made in a series of 25 counts. Although many of the differences were small, in some instances they were as great as 1,000 to 1,500 (10 per cent to 17 per cent). Furthermore, many investigators[8] have shown that there is a daily variation in the white blood cell count and a diurnal variation as well. Since the leukocyte count during the convalescent stage of rheumatic fever is usually not greater than 12,000 to 14,000, a single determination is considered to be unreliable. Nevertheless, if repeated counts are made over a period of time and the trend noted, the test becomes more valuable.

THE ERYTHROCYTE SEDIMENTATION RATE—There are numerous methods for determining the erythrocyte sedimentation rate. The one used in the present study is with minor modifications that devised by Rourke and Ernstene.[9] Repeated observations (usually every 5 minutes) over a period of one to one and one-half hours are made on a sample of heparinized blood in a graduated Wintrobe tube, and the maximum rate of fall of the red cells in millimeters per minute is determined. This is known as the sedimentation index. Since anemia in itself will increase the rate of fall, the tube is centrifuged, the hematocrit determined, and a correction made. The corrected value is called the corrected sedimentation index and is abbreviated C.S.I. The upper limit of normal for the corrected sedimentation index is 0.38 mm. per minute.

In contrast to the leukocyte count, the corrected sedimentation index is a more complicated test involving both time and a venipuncture. Nevertheless, when it is abnormal it is likely to be elevated far above the normal level so that it is more easily evaluated. This is obviously a distinct practical advantage. Daily unexplained variations of significant degree occur frequently enough to warrant check-up determinations before drawing definite conclusions.

CORRELATION OF CLINICAL SIGNS AND LABORATORY EVIDENCE OF ACTIVE RHEUMATIC FEVER

Of 121 successive admissions analyzed for clinical as well as laboratory signs of rheumatic fever, 73 (60 per cent) had clinical evidence of rheumatic fever, while 48 (40 per cent) had only laboratory signs of infection. The duration of these clinical signs of activity varied from a few days to as long as 12 months and averaged 3.4 months. The clinical manifestations observed in this series in their order of frequency were as follows: fever, nodules, chorea, epistaxis, joint pains, precordial pain, rash (usually the erythema marginatum type), congestive failure, abdominal pain, pneumonia, pericarditis, and pleurisy. The incidence and duration of these various signs have been summarized in table 1.

Table 2 summarizes the laboratory findings during the period of clinical manifestations.

TABLE 1

INCIDENCE AND DURATION OF THE CLINICAL SIGNS OF RHEUMATIC FEVER OBSERVED IN 73 PATIENTS

Sign	Case Incidence	Per Cent of Active Cases	Duration* Range	Duration* Average
1. Fever	39	53%	1 wk. to 12 mos.	2.4 mos.
2. Nodules	30	41%	3 wks. to 12 mos.	3.1 mos.
3. Chorea	21	29%	3 days to 4 mos.	3.2 wks.
4. Epistaxis	18	25%	1 wk. to 8 mos.	2.3 mos.
5. Joint Pains	17	23%	1 day to 2 mos.	2 wks.
6. Rash	14	19%	1 day to 6 mos.	2 mos.
7. Precordial Pain	14	19%	1 to 5 days	2 days
8. Congestive Failure	13	18%	2 wks. to 3 mos.	1 mo.
9. Abdominal Pain	10	14%	1 to 5 days	2 days
10. Pneumonia	7	9.5%	3 days to 2 wks.	1 wk.
11. Pericarditis	5	6.8%	3 days to 2 wks.	1 wk.
12. Pleurisy	4	5.4%	3 days to 2 wks.	1 wk.

*The duration noted is the time during which the symptoms were frequently, though not necessarily constantly, present.

TABLE 2

THE LEUKOCYTE COUNT (W. B. C.) AND CORRECTED SEDIMENTATION INDEX (C. S. I.) IN 73 PATIENTS WITH CLINICAL MANIFESTATIONS OF RHEUMATIC FEVER

	Both Tests Elevated	C. S. I. Elevated W. B. C. Normal	W. B. C. Elevated C. S. I. Normal	Both Tests Normal
Case Incidence	50	17	21	12
Average Duration	2.2 months	2.0 months	2.0 months	2.1 months

During the clinical course of the disease the status of some patients changed sufficiently necessitate their inclusion in the analysis two or more times. Thus it will be seen that otal of the four columns is 100 rather than 73.

This table represents an analysis of the clinically active group as a whole, irrespective of the severity of the illness. It will be seen that if there are clinical signs of active rheumatic fever, laboratory signs also are likely to be present. Not uncommonly, however, one or the other laboratory test may be normal, and occasionally both tests may be normal simultaneously in the presence of obvious clinical rheumatic fever.

For purposes of further discussion the clinical variations of rheumatic fever may be divided as follows: (1) severe rheumatic fever; (2) moderately severe rheumatic fever; (3) low-grade rheumatic fever. Although there were only 73 cases analyzed, the total of the following three groups is 97. This seeming discrepancy is the result of variations of rheumatic fever in some of the same patients during the period of observation.

Severe Rheumatic Fever — This group includes 24 patients, 14 of whom died. Fever was usually but not always present. More commonly the rectal temperature ranged between 101° and 102°. Rises to 103° were not uncommon, but fever of 104° to 105° was infrequent. The fever was often cyclic.

There was no correlation between the height of the fever and the severity of the infection, nor was there any correlation between the degree of elevation of the laboratory tests and the height of the temperature. When the patient was obviously acutely ill, the leukocyte count was in the majority of instances above 10,000 and usually well above this level. The corrected sedimentation index on the other hand, although also frequently quite high, was sometimes disproportionately low for the severity of the illness, and in some instances was even within normal limits.

Thus, of these 24 patients who were obviously acutely ill, the leukocyte count was consistently elevated well above normal in 16, while the corrected sedimentation index was consistently elevated in only 12 (50 per cent). When the leukocyte count was elevated, it was usually 14,000 to 16,000, but it was not infrequently higher than this and occasionally reached 20,000 to 30,000. An elevated corrected sedimentation index usually ranged between 0.7 mm. and 1.0 mm. per minute, but not infrequently was 1.1 to 1.4 mm. per minute, and reached as high as 1.9 per minute.

Westergren[10] has shown that congestive failure may lower the erythrocyte sedimentation rate under circumstances where one would expect it to be elevated. This may be the explanation for the frequency of low corrected sedimentation indices in this group of very ill patients, for of seven who had indices well within normal limits, five had frank congestive failure.

Congestive failure occurred only in patients who were relatively ill. In fact, in our experience, congestive failure in young patients has always been associated with other evidence of active rheumatic fever and therefore in itself should be considered a reliable sign of active infection. This is in accordance with the postmortem findings of Rothschild, Kugel, and Gross,[11] who found anatomic evidence of active rheumatic fever in the hearts of sixty out of sixty-one patients who died with congestive failure within the first two decades of life.

Moderately Severe Rheumatic Fever — There were 13 patients who at some stage of their illnesses appeared obviously but not dangerously ill. All of them had some degree of fever in addition to one or more other signs of rheumatic fever. The fever was usually cyclic, though in a few instances it was sustained. In general, the rectal temperature charts had peaks reaching 101° to 103°, but in several the level remained under 101°.

The leukocyte count was definitely elevated in 7 of these patients, ranging between 12,000 and 16,000. In the remaining 6 patients it fluctuated around 10,000 (8,000 to 13,000).

Of these 13 patients, 11 (84 per cent) had a consistently elevated corrected sedimentation index (0.6 to 1.4 mm. per minute), but 2 had periods when the corrected sedimentation index was normal in the face of obvious infection.

Low-Grade Rheumatic Fever — There were 60 patients who at some period did not appear to be ill and yet showed one or more clinical signs of rheumatic fever. Such low-grade rheumatic fever usually manifested itself by low-grade fever, nodules, rash, epistaxis, precordial pain, or mild joint pains, either individually or in combination. The rectal temperature elevations were usually between 100° and 101°. Where the fever was prolonged (several months or more) it was usually cyclic in nature. The term fever has been used to denote rises in rectal temperature above 100°.

In the majority of the cases with low-grade clinical rheumatic fever, one or both laboratory tests were elevated, with abnormal leukocyte counts ranging from 12,000 to 15,000, and abnormal corrected sedimentation indices varying from 0.5 to 1.0 mm. per minute. Normal laboratory tests (one or both) in the presence of clinical signs of infection were occasionally found. Thus, there were 12 patients in whom definite clinical signs of active infection continued in spite of a normal corrected sedimentation index. The case histories are briefly summarized, and the signs of active rheumatic fever shown during the period of a normal first I. The leukocyte count was also normal in to be 9 patients, but in the last 3 it was found elinelevated. In all 12 instances the

ical evidence of rheumatic heart disease on examination.

CASE 1. H. S., an 11 year old girl, for a period of 2 months had persistent nodules and occasional epistaxis. The P-R interval of the electrocardiogram was prolonged throughout this period.

CASE 2. F. S., a 16 year old boy, for a period of 1 month showed erythema marginatum almost daily, and had a prolongation of the P-R interval.

CASE 3. I. F., a 4 year old girl, had low-grade fever (up to 101°) and occasional epistaxis for 2 months.

CASE 4. C. B., a 7 year old girl, had persistent epistaxis as the only sign of active rheumatic fever for a period of 4 months. Packing and cauterization frequently were necessary to stop the bleeding, which occurred from the anterior septal region on either side. Local examination showed no underlying pathology to explain this. In the past, nosebleeds had been her only symptom of rheumatic fever and yet she developed rheumatic heart disease with aortic regurgitation.

CASE 5. W. B., an 11 year old boy, for 5 months had a persistent erythema marginatum rash and frequent rises in temperature to slightly over 100°.

CASE 6. B. H., an 11 year old girl, developed new nodules and for several days had joint pains at a period when the laboratory tests were normal.

CASE 7. R. D., a 13 year old girl, had chorea and nodules for a period of one month. For 3 days during this period she also had joint pains.

CASE 8. L. S., an 11 year old girl, had nodules for a period of one month.

CASE 9. S. T., a 7 year old boy, had chorea, low-grade fever, and daily erythema marginatum for 2 months.

CASE 10. L. K., a 13 year old girl, had joint pains over a period of several days. The leukocyte count was 14,000.

CASE 11. T. B., a 5 year old boy, had persistent cyclic low-grade fever (100.5°-101°) for 5 months. During the first 3 months of this period nodules were also present. The leukocyte count averaged 12,000.

CASE 12. F. B., a 15 year old boy, for one week had low-grade but definite fever. The leukocyte count averaged 12,000.

From the above it is evident that laboratory tests, although important, cannot be depended upon entirely for the recognition of low-grade rheumatic fever. A careful search for clinical signs must also be made.

E LEUKOCYTE COUNT AND CORRECTED SEDIMENTATION INDEX IN RHEUMATIC FEVER PATIENTS HOUT CLINICAL SIGNS OR SYMPTOMS

It subsi already been pointed out that after the of the clinical signs of rheumatic fever either an elevated leukocyte count or corrected sedimentation index, or both, usually persist. In about 40 per cent of the cases the clinical signs had already subsided by the time the patient was admitted to the hospital, but laboratory signs of infection were present. The duration of the laboratory evidence of active rheumatic fever varied from 2 weeks to 12 months. Patients were rarely kept in the hospital for longer than 12 months because of laboratory evidence of infection alone.

In a series of 163 rheumatic fever subjects it was possible to compare the duration of infection as evidenced by the leukocyte count and corrected sedimentation index. From table 3 it is seen that in 50 patients both tests returned to normal at about the same time. In 58 patients, the corrected sedimentation index remained elevated longer, while in 55 patients, the leukocyte count persisted longer at an elevated level. The length of time that any one test remained abnormal longer than the other in the majority of instances varied from 2 weeks to 4 months and averaged 2.1 months for the corrected sedimentation index and 2.4 months for the leukocyte count.

TABLE 3

COMPARISON OF THE LEUKOCYTE COUNT (W. B. C.) AND THE CORRECTED SEDIMENTATION INDEX (C. S. I.) AS TESTS FOR PERSISTENT ACTIVE INFECTION IN 163 RHEUMATIC FEVER SUBJECTS

	Persistent Elevation of W. B. C. in Presence of a Normal C. S. I.	Persistent Elevation of C. S. I. in Presence of a Normal W. B. C.	Simultaneous Return of Both Tests to Normal
Case Incidence	55	58	50
Per Cent	34.1%	35.5%	30.4%
Average Duration of Discrepancy	2.4 months	2.1 months	

Thus the leukocyte count and corrected sedimentation index are likely to indicate persistent infection with equal frequency. Since there is no constant correlation between the tests, one often being distinctly elevated when the other is well within normal limits, the determination of both tests is often necessary.

In most instances, after the subsidence of the clinical manifestations of rheumatic fever, abnormal laboratory findings return to normal within six months. Occasionally an elevated corrected sedimentation index or leukocyte count, or both, may persist without any associated clinical signs of infection. In such instances it has not always been possible to have the patient continue with bed rest for such a period. Upon discharge from the hospital they have been carefully observed in follow-up clinics, and a much restricted physical life is strongly urged. Some of these patients have had a recrudescence of clinically active rheumatic fever, but others have been followed for two years or more without any apparent change in their clinical or laboratory status. We believe that persistently ele-

vated laboratory tests in rheumatic fever subjects are significant, and that in the majority of instances they are indicative of persistent low-grade (subclinical) rheumatic fever.

The following four case reports illustrate such instances of prolonged abnormal laboratory findings.

CASE 1. A. C., House of the Good Samaritan, No. 5351, is a 12 year old male with a negative family history and past history. This patient had definite attacks of rheumatic fever in April and December, 1928, and chorea for 7 weeks in 1930. In March, 1931, he had a third attack of rheumatic fever with fever, swollen, tender joints, and epistaxis. He was seen at the Adolescent Cardiac Clinic of the Massachusetts General Hospital, and placed with the Children's Mission. Upon being transferred to the House of the Good Samaritan in June, 1931, he did not appear sick, and there were no clinical signs of rheumatic fever, but the leukocyte count was 19,000. His heart was borderline in size and there was a moderate mitral systolic murmur and a short mid-diastolic murmur. During August he had frequent low-grade fever (up to 100.8° R.) joint pains, and vomiting. The leukocyte count ranged between 15,000 and 19,000. He improved and remained asymptomatic in spite of a leukocyte count of 11,000 to 12,000 until October, when mild chorea appeared. The chorea, a borderline temperature (up to 100° R.), and an abnormal leukocyte count continued until June, 1932. During this period the corrected sedimentation index was normal. Thereafter there was again general improvement and gain in weight, but leukocytosis (10,000 to 14,000) persisted up to the time of discharge on October 30, 1932 (16 months after admission).

He did fairly well from October, 1932 to March, 1933, except for one cold in December. In March, 1933, he had another cold and sore throat which was followed by a recurrence of chorea, several nosebleeds, and loss of weight. He remained in bed at home until April 13, 1933, when he was admitted to the House of the Good Samaritan for the second time. Examination showed an ill-appearing boy with swollen, tender finger joints of the right hand, temperature of 101° (R.), and mild chorea. The cardiac findings were essentially the same as before, the leukocyte count was 17,000. On April 24 intravenous typhoid paratyphoid vaccine was given and was followed by an immediate frank rheumatic fever recrudescence with swollen, tender joints, fever of 103°-104° (R.), and a corrected sedimentation index of 1.4 mm. per minute. By the middle of May the fever, joint symptoms, and chorea had subsided and the corrected sedimentation index had returned to normal. There was good weight gain and improvement followed, but the leukocyte count remained elevated (11,000 to 13,000) up to discharge on November 22, 1933 (5 months after admission).

After discharge, follow-up examinations were made at the Massachusetts General Hospital, Adolescent Cardiac Clinic, at frequent intervals. He was soon found to be running a low-grade fever (100°-101° R.), a leukocytosis of 10,000 to 20,000, and he again lost weight. In February, 1934, mild chorea was observed.

On July 24, 1934, he was admitted to the House of the Good Samaritan for the third time because of persistent chorea. In addition to the chorea he was found to have a slightly elevated temperature (100.2° R.), a leukocyte count of 12,000, and corrected sedimentation index of 0.50 mm. per minute. The heart was not enlarged, a slight mitral systolic murmur was present but no diastolic murmurs could be made out. By August the chorea subsided, the temperature failed to rise above 100° (R.), and he had gained weight from 79 pounds to 102 pounds. The corrected sedimentation index returned to normal shortly after admission, but an elevated leukocyte count from 11,000 to 14,000 persisted. He was discharged on May 12, 1935 (10 months after admission).

Following discharge from the House of the Good Samaritan he was again seen frequently in the Adolescent Cardiac Clinic of the Massachusetts General Hospital. During this period he was fairly well except for several colds, occasional slightly elevated temperature, and continued leukocytosis. On December 28, 1935, he had a sore throat and several weeks later, during January, 1936, there was an acute onset of migratory, tender joints and fever of 105° P.O. By February he had again improved and he continued symptom-free thereafter until May, 1936, when he was again found to have mild chorea. The latter subsided by June, 1936. Throughout this period, however, the leukocyte count remained elevated. Associated with this last attack of rheumatic fever there was apparently an increase in the degree of cardiac involvement. The heart became slightly enlarged, the systolic murmur became louder, and there was an associated thrill, while at the apex there was a definite diastolic rumble.

CASE 2. A. S., House of the Good Samaritan, No. 5333, is a 15 year old male. His family history is negative, and his past history was irrelevant until 1927 (aged 6 years) when a routine school examination revealed the presence of heart disease. There had been no previous rheumatic symptoms. During 1928 he was examined from time to time at the Massachusetts General Hospital, Adolescent Cardiac Clinic. Because of several short periods of fever and failure to gain weight, he was admitted to the House of the Good Samaritan where he remained from June 15, 1931 to December 23, 1931. During this period of six months the only definite evidence of active infection was a persistently elevated leukocyte count which gradually fell from 17,500 to 11,500. The corrected sedimentation index was not determined. Cardiac examination revealed slight enlargement of the heart, moderate mitral systolic and short mitral mid-diastolic murmurs, and a slight, rough aortic systolic murmur.

During 1932 he had several upper respiratory infections, lost weight, and continued to run a mild leukocytosis (10,000 to 15,000). Therefore, he was readmitted to the House of the Good Samaritan on October 21, 1932 and remained until January 1, 1933. During this period an elevated leukocyte count (11,000 to 15,000) and a failure to gain weight was again the only definite evidence of active infection. The corrected sedimentation index remained within normal limits during this period. The cardiac findings were essentially as before.

From early in 1933 to the end of 1934 observations at the Massachusetts General Hospital, Adolescent Cardiac Clinic, revealed gradual improvement in appearance, gain in weight from 58 to 68 pounds, and a constantly normal corrected sedimentation index. However, the leukocyte count continued to fluctuate between 10,500 and 15,500. During 1935 his status remained essentially the same except for four upper respiratory infections.

Early in January, 1936, there was a frank rheumatic fever recrudescence which was ushered in by one to two nosebleeds daily. Two weeks later his face became puffy and his urine very dark in color. He gained 12 pounds in less than 3 weeks. On January 28, 1936, he was admitted to the Massachusetts General Hospital where he was found to appear moderately ill. There was edema of the face

and the liver edge was felt three fingerbreadths below the costal margin. The heart had become larger, the apex impulse being felt in the anterior axillary line. The rate was rapid and there were moderately loud mitral systolic and diastolic murmurs. There was no fever, but the corrected sedimentation index was elevated (0.81 mm. per minute), and the leukocyte count was 12,000. The urine contained a large trace of albumin and many red blood cells. Its specific gravity was 1.024. Blood N.P.N. was 43 mg. per cent, and the plasma protein 6.4 mg. per cent.

The leukocyte count fluctuated between 12,000 and 20,000. On February 9, a pleural friction rub was heard and associated with this there was a temperature of 102°. The fever subsided after 4 days, and thereafter the patient gradually improved. He was discharged from the hospital to a convalescent home on March 13, 1936, at which time renal function tests were normal and the urine was negative except for a few red blood cells. The leukocyte count was 14,000.

CASE 3. E. S., House of the Good Samaritan, No. 5491, a 23 year old single female with negative past history and irrelevant family history, had rheumatic fever for the first time at the age of 12 (1921). Following this she remained well, but a heart murmur was noted when she was 17 years old. Nevertheless, she continued symptom-free until 1930 (aged 20) when associated with a protracted cold she became tired and dyspneic. Her physician prescribed digitalis and bed rest which gave only temporary improvement. In the fall of 1931 the dyspnea increased, she became nauseated and vomited, and her legs became swollen. In January, 1932, she was admitted to the Massachusetts General Hospital, where she was found to have rheumatic heart disease and generalized anasarca. Treatment with digitalis, diuretics, and Southey tubes caused only partial improvement in the congestive failure. During this period frequent low-grade fever and a transient pericardial friction rub gave evidence of active rheumatic fever.

She was transferred to the House of the Good Samaritan on May 6, 1932, approximately 9 months after the onset of the congestive failure. Physical examination revealed an ill-appearing young woman with orthopnea, moderate engorgement of neck veins, and edema of legs and sacrum. The heart was considerably enlarged with a faint apical systolic murmur and a well-marked mitral diastolic murmur with a presystolic crescendo accentuation. The rhythm was regular.

With long bed care, digitalis, and diuretics the patient gradually improved and became apparently edema-free by September, 1932. Despite the disappearance of congestive failure as a clinical manifestation of active rheumatic fever, the P-R interval of the electrocardiogram was frequently prolonged (ranging from 0.19 to 0.23 seconds), and the corrected sedimentation index was moderately elevated (0.5 to 0.8 mm. per minute). The former was possibly related to digitalis therapy, but the latter could be explained only on the basis of active rheumatic fever. At no time did she have any joint pains, nosebleeds, nodules, rash, or fever. The leukocyte count was always well within normal limits.

In February, 1933 (9 months after admission to the House of the Good Samaritan), without any obvious precipitating cause except perhaps excitement, she suddenly developed auricular fibrillation, rapidly went into severe cardiac failure, and died within 24 hours.

A postmortem examination revealed definite microscopic changes in the heart indicative of active rheumatic fever.

CASE 4. E. F., House of the Good Samaritan, No. 4002, a 16 year old female with a negative family history and an irrelevant past history, had rheumatic fever for the first time in 1924 (aged 6 years). In April, 1925, active rheumatic fever was manifested by nosebleeds and easy fatigue. She was admitted to the Boston Dispensary and on June 5, 1925, transferred to the House of the Good Samaritan. Her heart was slightly enlarged and there were mitral systolic and diastolic murmurs. Observation for 2 months revealed no clinical or laboratory evidence of active infection, and on August 7, 1925 she was discharged.

Check-up examinations were made at the Boston Dispensary from 1925 to 1933. During this period she had frequent colds and four mild recurrences of rheumatic fever in 1929, March, 1930, April, 1931, and November, 1931.

In January, 1933 there was a frank recrudescence of rheumatic fever with fever and joint pains and on February 8, 1933 she was admitted to the House of the Good Samaritan for the second time. Examination revealed that since 1925 there had been a marked advance in the cardiac involvement. The heart was considerably enlarged, and there was associated the signs of mitral stenosis and aortic regurgitation.

She remained at this hospital for 17 months and throughout this period there was a prolongation of the P-R interval of the electrocardiogram and frequent exhibitions of erythema marginatum rash. The corrected sedimentation index was usually elevated (0.5 to 1.2 mm. per minute), but the leukocyte count was always under 10,000. In addition to these findings there were the following significant episodes:

In March, 1933, there was a bout of fever (101°-102°) for one week, and several nosebleeds. On April 24, 1933, intravenous typhoid paratyphoid vaccine was followed by fever for 2 days and several nosebleeds. In September, 1933, there was again slight fever for 1 week. From November to December, 1933, the patient had occasional joint pains. On February 15, 1934, intravenous typhoid paratyphoid vaccine was again given and was followed by a definite increase in the P-R interval of the electrocardiogram, in addition to fever and joint pains for a few days. After the subsidence of this mild recurrence, the corrected sedimentation index remained abnormal, and the P-R interval prolonged. On July 1, 1934, without evident cause, the patient had a severe chill. The temperature rose rapidly to 105° (R.), and was followed by severe pulmonary edema. All attempts at therapy were to no avail. She rapidly became worse and died within 8 hours after the chill.

Postmortem examination demonstrated abundant evidence of active rheumatic fever.

The above four case reports give ample evidence of the chronicity of rheumatic fever and the frequency of recurrences. Further, they indicate that repeated abnormal laboratory findings, even in the absence of clinical signs or symptoms of rheumatic fever, must be interpreted as evidence of active rheumatic fever (elevated leukocyte count in cases 1 and 2, fast corrected sedimentation index in cases 3 and 4). It has been our clinical experience that patients showing persistently abnormal laboratory findings are peculiarly liable to recurrences of clinical rheumatic fever. In cases 3 and 4, death resulted too quickly for those pathologic changes to develop which are seen commonly in pro-

tracted rheumatic fever. Despite this, such changes were found at postmortem. The presence of subclinical rheumatic fever is thus demonstrated, as well as the importance of abnormal laboratory tests during such periods.

ELECTROCARDIOGRAPHIC EVIDENCE OF ACTIVE RHEUMATIC FEVER

Of 121 patients with active rheumatic fever, there were 14 who at some time during the course of their illness showed a prolonged auriculoventricular conduction time as measured by the P-R interval of the electrocardiogram. Six of these had a normal corrected sedimentation index. Four of these 6 had other signs of rheumatic fever in addition to the long P-R interval, but in two patients the long P-R interval was the only sign of active rheumatic fever.

Thus, the electrocardiogram may present confirmatory evidence of the continuance of active rheumatic fever, and occasionally may be the only indication of the activity of the process.

OTHER FACTORS AFFECTING THE CORRECTED SEDIMENTATION INDEX

The interpretation of the corrected sedimentation index must be made with caution, for it is an extremely sensitive test. It is not specific for rheumatic fever, and may be elevated by tuberculosis, renal infection, and many other diseases. Perhaps less well known, however, is the fact that seemingly minor events may cause the corrected sedimentation index to become and remain elevated for as long as two to three weeks. Two such events may be mentioned.

Upper Respiratory Infections — Westergren[10] observed the effect of tonsillitis on the sedimentation rate in three children with increased hilus glands and positive tuberculin tests, but no other evidence of active tuberculosis. The sedimentation rate was definitely elevated by the second day, but did not reach a maximum until the temperature was already on the decline. A normal level was not again reached until two to four weeks later. Bortree[12] likewise found that in normal individuals simple colds caused the sedimentation rate to become increased and remain rapid for four days to two weeks.

Our observations on the effect of sore throats (tonsillitis or pharyngitis) on the corrected sedimentation index in rheumatic fever subjects are confirmatory. In convalescent rheumatic fever patients with normal indices, the sore throat was followed by elevated rates for as long as three weeks after the patient was apparently completely recovered. Cervical adenitis and other complications caused an abnormal level to continue even longer.

Tonsillectomy—Löhr[13] reported observations on the sedimentation rate in 50 patients with healing sterile wounds. Postoperatively the sedimentation rate rose as early as twelve hours and was always definitely above normal by twenty-four hours. Return toward normal did not begin until six to ten days later.

We have recorded in figure 1 the effect of tonsillectomy on the corrected sedimentation index in eight rheumatic fever subjects. These patients were all in the convalescent stage of their disease, and at the time of the operation showed no evidence of active infection. The postoperative course was uneventful in all. It is seen from the chart that the corrected sedimentation index rose promptly after the operative procedure, and that a return to the preoperative level in many instances was delayed for as long as two weeks.

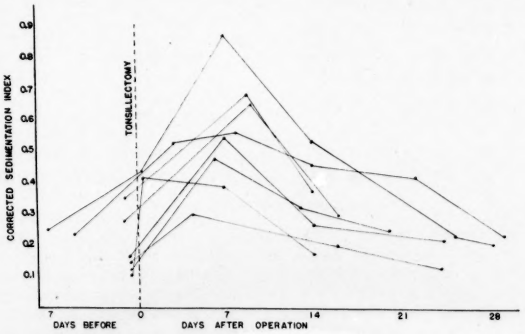

FIGURE 1. The effect of tonsillectomy on the corrected sedimentation index in eight rheumatic fever subjects.

SUMMARY AND CONCLUSIONS

1. A clinical and laboratory study of 178 rheumatic fever subjects is presented.

2. The leukocyte count and corrected sedimentation index are helpful and often essential for the determination of low-grade rheumatic fever. In the majority of instances, after the clinical manifestations of the disease subsided, one or both of these tests continued at an elevated level for several weeks to many months.

3. As tests for low-grade rheumatic fever, the corrected sedimentation index and leukocyte count are of about equal value. In view of the large percentage of error in the technic of the leucocyte count, and the necessity for repeated counts, the corrected sedimentation index is more valuable as a single, isolated test.

4. One or both tests may be normal in the presence of clinical signs of rheumatic fever.

5. Corrected sedimentation index determinations are of no significance in the evaluation of active rheumatic fever if performed within two or three weeks after an upper respiratory infection or tonsillectomy.

6. Neither the leucocyte count nor the corrected sedimentation index is a specific rheumatic fever test, and must be interpreted with regard to other considerations.

7. In rheumatic fever subjects, repeatedly elevated leukocyte counts and rapid corrected sedimentation indices should be considered indicative of subclinical rheumatic fever in the absence of other cause for their abnormality.

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CARCINOMA OF THE BREAST IN YOUNG WOMEN

BY GRANTLEY W. TAYLOR, M.D.*

THERE is fairly general agreement in the literature that carcinoma of the breast in young women differs in many respects from carcinoma of the breast in older women. The recent studies of Nathanson and Welch[1] demonstrate the effect of age on life expectancy in this disease, and the graver prognosis in younger women. The gravity of prognosis in carcinoma of the breast associated with pregnancy or lactation has received considerable attention.[2, 3, 4, 5] Inflammatory carcinoma of the breast[6] with its poor prognosis is largely confined to a young age group. Trout[7] has called attention to the dangers of a subsequent pregnancy following apparent cure of the disease. Other evidence of difference in behavior of the disease has recently been brought forward by Dresser[8] and Martin,[9] who have demonstrated regression of established bone metastases following irradiation of the ovaries in a certain proportion of cases in cancer of the breast in young women.

The present study was undertaken to analyze further the causes of the difference in prognosis which have been observed. The cases of carcinoma of the breast observed at the Massachusetts General Hospital during the years 1921 to 1929 have already been presented in end-result studies.[10, 11, 12] These cases have been

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divided into three age groups, 146 cases 45 years or younger, 303 cases aged 46 to 60 inclusive, and 156 cases over 60. The study is confined to the youngest and oldest groups, and roughly it may be said that the youngest group contains only women who have not yet passed the menopause, while those in the older group have certainly done so.

Although data on preoperative duration of carcinoma are unreliable, it was found that the younger women reported for examination considerably earlier than did the older women, when cases of comparable extent were considered. Thus when the disease was confined to the breast, without axillary involvement, three quarters of the younger group reported an average duration of less than six months, while only half of the older women recorded a similar brief duration.

The two groups were compared in regard to the presence or absence of axillary lymph node metastasis at the time of first examination. The results are shown in table 1. It is evident that 77 per cent of the younger group present axillary involvement when first seen, while only 57 per cent of the older women present a comparable degree of extension. It is fair to say that carcinoma of the breast tends to metastasize to the axillary lymph nodes earlier in young women.

TABLE 1

AXILLARY INVOLVEMENT ON ADMISSION

Age	Under 46	Over 60
Axilla not involved	23%	43%
Axilla involved	77%	57%

The groups were then compared in regard to the grade of malignancy, as shown by histologic study.[12] The result of such a comparison is shown in table 2. From this table it is apparent that about 16 per cent of the older women present cancers of the lowest grade of malignancy, while only 28 per cent are highly malignant. This is in marked contrast to the younger group, in which only 7 per cent are of the lowest grade, while 44 per cent present highly malignant growths. It is evident that, in general, cancer of the breast is likely to be of higher grade of malignancy in younger women than in older women.

TABLE 2

PATHOLOGIC GRADE OF MALIGNANCY

Age	Under 46	Over 60
Cases	146	156
Grade I	7%	16%
Grade II	49%	56%
Grade III	44%	28%

Five-year cures were secured in 34 per cent of the younger group, and in 44 per cent of the older group (table 3). This difference is adequately accounted for by the differences in the two groups as regards axillary involvement and as regards the grade of malignancy. When comparison is made between disease of equal extent, or of equal grade of malignancy in the two groups, the differences disappear. Thus cures in cases without axillary involvement in the young group were 71 per cent, in the old group 67 per cent. Cures in cases with axillary involvement were 20 per cent and 21 per cent respectively. Similar comparison can be made between the two groups for cases of comparable grades of malignancy. Although the numbers of cases become too small to permit percentage comparison, it is evident that the age groups show no essential difference in the curability of comparable grades of malignancy of comparable extent.

TABLE 3

CURES WITH RADICAL OPERATIONS

Age	Under 46	Over 60
Cases	117	113
Cures	34%	44%
Cures—Axilla not involved	71%	67%
Cures—Axilla involved	20%	21%

The two age groups may be contrasted as regards the promptness of recurrence following radical operation (chart 1). In cases with axillary involvement one year after operation, 95 per cent of the older women still survive, as compared with 75 per cent of the younger group. Eighteen months after operation the figures are 80 per cent and 60 per cent respectively. The disparity diminishes later, and after 30 months the two groups are essentially alike.

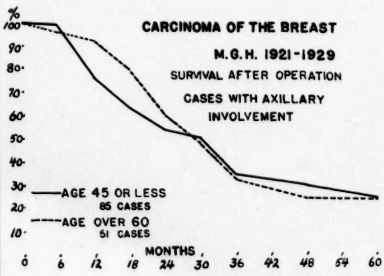

CHART 1.

A similar chart for cases without axillary

involvement at the time of operation shows no essential difference between the two age groups.

DISCUSSION

On the basis of observed regressions of established metastases following ovarian radiation, it has been proposed[14] that radiation artificial menopause be brought about postoperatively in young women as a prophylactic against recurrence. In the light of our analysis of this series of cases, it is difficult to see how such a procedure would improve our present results of operation. The difference in five-year cures between the two age groups is adequately explained by the greater number of patients with highly malignant growths among the younger women, and the greater number of cases with established axillary metastasis at the time of operation. There is no evidence that ablation of ovarian function can modify the histologic characteristics of a malignant growth in the breast; nor can postoperative ovarian radiation affect the percentage incidence of axillary involvement at operation.

The difference in behavior of the two age groups as regards promptness of recurrence following operation, as shown in chart 1, might well be modified or diminished by postoperative artificial menopause. Apparently, the greatest benefit that could be hoped for would be to raise the curve of the younger group to that of the older group. It has been pointed out, however, that the difference between the two curves is evident only during the first few years, and that thereafter the two groups behave alike.

It is inevitable that a study such as this, which is concerned with operable carcinoma of the breast, cannot include a fair sample of the disease problem, especially as regards the younger group of patients. Figures as to operability are subject to wide variation, depending on the character of the hospital, the class of population which is served, and numerous other factors. It is likely that a larger percentage of cancers of the breast in young women are inoperable when first examined. This fact would be suggested by the more rapid rate of growth and the tendency to earlier metastasis observed in our series. Our series also does not include any cases complicated by pregnancy or lactation, or any cases of the inflammatory type. Instances of this sort emphasize the intimate relation of certain carcinomas of the breast to hormonal influences. Finally, our series can shed no light on the mechanism of the regressions of metastases after ovarian radiation. It is especially these regressions which have justified the recommendation of postoperative prophylactic artificial menopause. Histologic study of areas of regression is greatly needed. It may be that this will provide evidence that the histologic characteristics of the disease may be modified by ovarian radiation.

CONCLUSIONS

Carcinoma of the breast tends to metastasize earlier in young women than in older women.

Carcinoma of the breast tends to be of higher grade of malignancy in younger women.

Postoperative recurrence takes place more promptly in younger women.

In carcinomas of the breast of equivalent extent, and of equal grades of malignancy, age alone does not affect the curability.

There seems to be no ground for the inference that routine postoperative radiation of the ovaries would have modified appreciably the results observed in the younger age group.

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THE ROLE OF SCURVY IN THE ETIOLOGY OF CHRONIC SUBDURAL HEMATOMA*

BY THEODORE HUNT INGALLS, M.D.†

THE purpose of this paper is to record certain suggestive evidence of the role which scurvy or a deficiency of vitamin C may play in the production of chronic subdural hematoma.‡ Because of the recent impetus given to the study of scurvy by quantitative chemical analyses of ascorbic acid, in the tissues, blood, and urine;[27] by the elucidation of its basic pathologic changes;[24, 18] and by its earlier x-ray recognition in infancy[28] it appears that this is an opportune time for the publication of a conception evolved from a study of the subject during the past three years.

That chronic subdural hemorrhage is associated with poor hygiene and disturbance of nutrition in one form or another, has long been recognized. Thus Doehle[1] noted the presence of rickets in at least 13 of 57 cases, and Rosenberg[2, 3] after long experience with the disease stated he had never seen it occur in a healthy, normally developed, breast-fed child. Sherwood[4] reported from The Infants' and The Children's Hospital in Boston, nine cases in 1930 and pointed out that five of the patients were cared for in institutions or by foster mothers.

In reviewing the histories of these cases, attention was attracted to the possibility that scurvy may have been the underlying etiologic factor. Since x-ray studies of the long bones had been made in but five of the nine infants, those particular cases were selected for detailed study; the results are tabulated in table 1.

Of these five cases with chronic subdural hemorrhage, three were found to have x-ray evidence of scorbutic changes in the long bones. In the other two cases the history and examination were suggestive of scurvy, though the x-rays exhibited no positive findings.

Hess and Unger[5] have said, "It is evident that the fact that beading of the ribs may be of scorbutic origin has clinical and diagnostic significance. Unless we are certain that a baby has received an adequate quantity of antiscorbutic food, we are not justified in considering enlargement of the costochondral junctions of rachitic origin. The rosary may indeed be of two-fold nature, as these two nutritional disorders may, and frequently do, exist concomitantly." In the two infants without x-ray changes in the long bones, both exhibited costochondral changes and it was fairly certain that they had not received an adequate quantity of antiscorbutic food.

A tabulated comparison of the positive findings in the last two series of cases, reported by Peet and Kahn, and by Sherwood, reveals a strikingly poor social and nutritional background (table 2). Eleven of the eighteen patients were either illegitimate or had been cared for in foster homes; ten were specifically recorded as being artificially fed; and only one was stated to be breast-fed. It is of interest to note that in the case occurring in a breast-fed baby the onset was at thirteen months of age, the latest in both groups. The disease did not occur until the breast feeding had been stopped.

Instances of subdural hemorrhage associated with acute scurvy are not wanting. Gilman and Tanzer[6] have reported a case in a boy of sixteen months who was breast-fed for two months, and then weaned on cow's milk with an inadequate supplement of orange juice. He was operated upon, a clot extracted, and recovery ensued. These authors reviewed cases of a similar condition in infants reported by Sammis,[7] Ord,[8] Meyer,[9] Hayem,[10] and Sutherland.[11] Sutherland's two cases in 1893 are extremely interesting. They were girls with advanced scurvy, aged two years, and fourteen months, respectively. At autopsy each was found to have extensive subdural hemorrhage, in addition to which one had a soft, red, gelatinous material in the spinal canal just beneath the dura, and the other had two subcortical hemorrhages. He concluded that these hemorrhagic lesions were the same as Virchow's pachymeningitis interna hemorrhagica because there was: (1) a tendency to recurrent attacks of hemorrhage without local inflammation; (2) coagulation and organization of the extravasated blood; and (3) the effects were entirely those of mechanical pressure. He emphasized that "these conditions are also characteristic of the subperiosteal and other hemorrhages of scurvy".

In addition to these instances the American Pediatric Society's report[12] on infantile scurvy in 1898 set forward three of twenty-nine deaths as due to cerebral hemorrhage. The diagnoses were made clinically. Stokes and Campbell[13] reported three fatal cases of infantile scurvy, in one of which subdural hemorrhages were

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‡The term chronic subdural hematoma as used in these pages is synonymous with pachymeningitis interna hemorrhagica of the older writers.

†Ingalls, Theodore Hunt—William Hunter Workman Fellow of Harvard University Medical School. For record and address of author see "This Week's Issue," page 1313.

found at autopsy. Hess[14] in 1914 stated, "Finkelstein mentions that hemorrhage (in infantile scurvy) has been found postmortem in the dura mater. In one of our cases in which convulsions supervened in the course of the disease, the blood-tinged cerebrospinal fluid led us to believe that we were dealing with a case of this nature." This is interesting and pertinent, inasmuch as convulsions and bloody cerebrospinal fluid are the most constant findings in chronic subdural hemorrhage; if the clinical signs of advanced scurvy had been overlooked, this case would doubtless have been diagnosed chronic subdural hemorrhage.

DISCUSSION

In studying the incidence of chronic subdural hemorrhage as reported by various authors, it is at once striking that it occurs primarily in infants, chronic alcoholics, and the insane.[1, 2, 3, 15, 16, 17]

The particular advantage of studying the entity of chronic subdural hemorrhage in infancy is the relative ease with which the nutritional aspects can be evaluated and the fact that vitamin C deficiency is manifested in the x-ray by pathognomonic bone changes which when present to a sufficiently advanced degree give conclusive objective evidence of the dietary error. We now know that subclinical scurvy may exist before clinical manifestations and x-ray changes become apparent.

In the group of nine infants studied at The Infants' and The Children's Hospital in Boston all were bottle-fed. Case 9 is excluded from this discussion, as it was an instance of birth hemorrhage. Five of the eight remaining patients were cared for in institutions or by foster mothers, and six were specifically recorded to have had rosaries of varying degrees. X-rays of the long bones were available for study in five of these cases, and in three roentgenologic evidence of scurvy was elicited.

It is not to be wondered at that active scurvy could not readily be demonstrated in each patient in this series, since the essence of the conception here set forward, is that of a chronic low-grade process. One might say that chronic subdural hemorrhage as encountered clinically and at autopsy is an end stage, far removed from the initial series of events. The scorbutic process itself may have been arrested, without obviating the effects of repeated cerebral hemorrhage. It is not unreasonable to assume that close scrutiny of the dietary of the alcoholic and the insane would often reveal deficiency of vitamin C, a finding so much more subject to objective proof in early life. Ascorbic acid levels in the blood, and urine studies for the determination of the degree of saturation with the vitamin, offer new methods of investigating latent scurvy.

That these three groups of individuals are subject to relatively frequent head trauma is an observation that needs no carefully adduced evidence. It is more pertinent to point out that histories of such patients are often of necessity inaccurate, and completely obscured in this respect. That a smaller increment of trauma is necessary to produce bleeding when added to a "hemorrhagic diathesis" is also obvious.

In chronic subdural hemorrhage the source of the bleeding is usually regarded as a seepage following the rupture of "bridging veins" between the pia and dura mater.[17, 19] Another plausible explanation could be hemorrhage from minute bleeding points where arachnoid villi were torn away at their invaginations into the dural sinuses. In vitamin C deficiency with its consequent resorption of intercellular substance, the security of the arachnoid villi must suffer along with other structures dependent upon connective tissue support. Wolbach[20] has observed, "The arachnoid villi because of their structure and relationship to compressible veins and sinuses furnish yielding points in the enclosement of the brain," and "form the weakest points in the dura".

The fate of blood in the subdural space in healthy individuals has been investigated by Van Vleuten,[21] Boeckmann,[22] and Putnam and Putnam.[23] These workers were agreed that, in the natural course of events, subdural blood was subject to resorption and organization. Putnam and Putnam state that "the subdural clots in our cases were always small and never showed evidence of progressive or repeated hemorrhage". These were substantially the same findings they obtained in experimental animals. This is not the state of affairs in chronic subdural hematoma where there is a progressive series of hemorrhages, without effective organization. Some factor not already considered in previous clinical or experimental conceptions of chronic subdural hemorrhage must be of importance. That the factor may be scurvy is indicated by the clinical, sociologic and epidemiologic considerations enumerated above.

Wolbach[24] has stressed the importance of vitamin C in repair. Thus the very condition which might well account for easy bleeding into the subdural space, would also be the one in which organization and resorption of extravasated blood would be most difficult. This is in accord with the progressive nature of chronic subdural hematoma. In his study on the formation of collagen and reticulum, Wolbach[25] has shown that repair of the blood clot in absolute scorbutus begins promptly by the migration of fibroblasts from adjacent tissues into the clot and the continued division of these cells. Capillaries do not penetrate the clot for any considerable distance, and although closed columns of endothelial cells grow into it they are apparently unable, due to

lack of intercellular substance, to form functioning capillaries. Such a situation could serve to give rise to a fibrous vascular zone, in short a neomembrane, without adequate organization of the interior. Progressive hemorrhages and progressive remissions and exacerbations of a scorbutic process could account for all the variations of the velvety membrane found at autopsy. Zollinger and Gross[19] have demonstrated that this structure has the properties of a semipermeable membrane. It is well known that encysted collections of erythrocytes may remain intact for a long period of time, and that it may be a matter of months before complete disintegration of red cells with liberation of free hemoglobin from their stroma is gradually accomplished. These workers found the osmotic pressure of whole blood one-fifth that of hemolyzed blood. Furthermore, since osmotic pressure is directly proportional to the molecular concentration of a fluid, they concluded that "disintegration of hemoglobin to a certain point, is attended by an enormous rise in osmotic tension". Such a conception in their opinion could account for the slow addition of fluid and enlargement of the hematoma sac to two or three times its original size. The slow augmentation in size would account for the late onset of symptoms in this condition.

SUMMARY

Chronic subdural hematoma occurs primarily in the infant, the alcoholic, and the insane. On several occasions subdural hemorrhage has been conclusively demonstrated as a complication of scorbutus. A closer analysis of five of the "idiopathic" cases of chronic subdural hemorrhage observed in this hospital has been attempted. In three of them, x-ray evidence of scorbutic bone changes was found. The epidemiologic and sociologic implications of existing data in this regard have been emphasized.

The following sequence of events seems to be concerned with the production of idiopathic chronic subdural hematoma: an underlying bleeding diathesis is postulated in most cases on the basis of scurvy. Free subdural bleeding is produced by a definite or insignificant head trauma causing either disruption of arachnoid invaginations into the dural sinuses, or rupture of a bridging vein. Organization and removal of the clot are impeded by the scorbutic process resulting in a neomembrane surrounding free blood. Repeated trauma and remission and exacerbation of the scurvy modify the disease. Late symptomatology is occasioned by enlargement of the semipermeable sac following the slow breakdown of red blood cells and reduction of free hemoglobin into smaller molecular aggregates with the passage of time.

CONCLUSION

The role of scurvy in the etiology of chronic subdural hematoma is emphasized.

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TABLE 1

Case No. in Sherwood's Series	Age, Date and Chief Complaint	History	Present Illness	Examination	Laboratory Studies	X-Rays Original Findings	X-Rays Review Comment‡
1.	E. F. 4½ mos. C. C. Crying as though going into convulsion. Sept., 1928.	P. H. Sent to Boarding Home at 2 wks. Not breast-fed. No C. L. O.* or O. J.†	P. I. Suddenly became fussy, irritable and screamed. In half an hour more had a convulsion.	P. E. Head: 16 inches. Not irritable. Fundi: Many hemorrhages. Chest: Sl. tendency toward rickets. Feet and legs cold and blue.	Spinal tap clear. Subdural: Bloody and xanthochromic. Pressure elevated.	Films of extremities except for sl. rachitic changes are essentially negative.	Slight osteoporosis. Few small clefts in radius; lines of arrested growth. Inconclusive.
2.	C. R. 11 mos. C. C. Loss of weight. Vomiting 3 wks. April, 1929.	P. H. Mother unmarried. Baby boarded out. Never breast-fed. C. L. O. and O. J. amounts not known.	P. I. 3 wks. ago had a convulsion with vomiting since. Has been more irritable than ordinary.	P. E. Irritable and poorly nourished. Head: 18⅝ inches. Eyes: Displaced downward. Fundi: Right, sl. blurred about disc margin. Veins congested. Chest: Sl. tendency to funnel shape. C. C. junctions enlarged. Long Bones: Sl. bowed.	Bloody S. F. Xanthochromic. Pressure elevated.	Skull: Marked separation of sutures. Chest shows rather marked clubbing of ribs. Extremities: Marked generalized osteoporosis due evidently to chronic malnutrition. No evidence of recent rickets.	"Ground glass" appearance of shaft, pencilling of cortex, halo-like epiphyses and metaphyseal bands. Scurvy.
3.	C. E. 7 mos. C. C. Enlarged head. Convulsions. Sept., 1927.	P. H. Never breast-fed. No mention of O. J. Always sick and colicky. Has never slept well—cries most of day. Vomitus slime 2½ mos.	P. I. 3 wks. ago had a convulsion and 3 days ago a second. Head is getting bigger. Cries most of day.	P. E. Head: 18 inches. Fundi: Marked hemorrhages. Chest: Sl. enlargement of C. C. junctions. Liver: 2 fingers.	Bloody L. P. Xanthochromic. Likewise subdural fluid. Pressure elevated.	Skull: Some separation of sutures. Long bones show no definite variation from normal.	Flaring of the rib ends and increased density. Not recognizable, however, as due to scurvy.
5.	E. B. 9 mos. C. C. Something in brain. Oct., 1928.	P. H. After birth baby boarded out—it has been in 3 homes, one of which was very filthy. Father and mother will not care for it.	P. I. 2 mos. ago had convulsions. Have been recurrent since.	P. E. Quite irritable when awakened. Small punctate wound on scalp. 7 to 8 similar lesions on 4th lumbar vertebra. Some evidence of tenderness over it. Minute petechial spots seen in R. retina. Head: 17¼ inches.	Subdural tap bloody and xanthochromic. Pressure elevated.	Marked separation of sutures, evidently due to increase of intracranial pressure, etiology of which is not evident. Old fracture of middle third of rt. radius. Bones otherwise essentially negative.	Definite cleft in distal metaphysis of rt. ulna. Long bones show lines of arrested growth, halo-like epiphyses, and dense bands at metaphyseal margins. Consistent with scurvy.
6.	A. A. 10 mos. C. C. Unusual enlargement of head—2 wks. Dec., 1928.	P. H. Never breast-fed. Good C. L. O. and O. J. history. General health always poor.	P. I. 5 mos. ago—vomiting 1-2 a wk. 3 mos. ago—projectile. 2 wks. ago—large head. Baby very irritable — utters shriek when either shoulder is touched.	P. E. Scanty. Comatose. Large head. Eyes: Displaced downward.	Sl. bloody and xanthochromic fluid. Colon B. cultured. Wass.: ? False positive.	Marked separation of sutures. Extremities show altered density near ends of long bones. Epiphyses are halo-like. Pt. has evidently had a scurvy which has now practically healed.	Plates have been lost. Comment is taken from record.

*Cod liver oil.
†Orange juice.

TABLE 2

		Home	Birth	Feeding	History	Onset
Peet and Kahn's[28] Series (1932)	I	Illegitimate	F. T., N. D.	On a formula at 10 days.		4 mos. Large head; no convulsions.
	II	Illegitimate		Formula almost immediately.	After birth "discharged to nursing home as normal".	3 mos. Apathy; not moving lt. arm.
	III		N. D.	Had always been on a formula.		5½ mos. Project. vomiting, convulsions.
	IV		N. D.	On a formula at 2 weeks.		4 mos. Enlarged head, convulsions.
	V	Illegitimate	N. D.	Soon placed on a formula.		3 mos. Enlarged head, convulsions.
	VI		Inst. Del.	Breast-fed for 10 months.	Mother Negro with positive Wass.	13 mos. Past 2 mos. appetite poor, irritable, vomiting.
	VII		7 mos. N. D.		At 4 mos. losing wt. Loss of appetite and diarrhea.	10 mos. Enlarging head.
	VIII	Illegitimate			Discharged from maternity home at 6 wks. Good condition.	6 mos. Enlarging head.
	IX	Illegitimate				Enlarging head.
Sherwood's Series (1930)	I	Boarding Home		Not breast-fed. No C. L. O. or O. J.	Mother had had syphilis and received some treatment.	4½ mos. Irritability. Convulsions.
	II	Illegitimate	F. T., N. D.	Never breast-fed. C. L. O. and O. J. not known.	Mother unmarried, child boarded out.	10 mos. Loss of wt., vomiting.
	III		F. T., I. D.	Never breast-fed.		6 mos. Convulsions, enlarging head.
	IV	Charitable Institution	N. D.		Had had rickets.	7 mos. Convulsions.
	V	Boarded Out	N. D.		Father and mother refused to care for baby. It had been in three homes.	7 mos. Convulsions.
	VI	Boarded Out	N. D.	Never breast-fed.		6 mos. Vomiting, enlarging head.
	VII	Foster Mother	N. D.		Mother dead.	5 mos. Convulsions.
	VIII		N. D.			10 mos. Vomiting.
	IX		F. T., ? D.		Cyanotic first 2 days of life. (Diagnosed as birth hemorrhage.)	3 wks. Vomiting, large head, convulsions.

‡The writer is indebted to Dr. E. C. Vogt of The Children's Hospital, Boston, and to Miss Deborah Jackson of the Harriet Lane Home, Johns Hopkins Hospital, for their discussion of these plates.

BRUCELLOSIS (UNDULANT FEVER)

Interesting and Important Facts About the Disease With the Report of a Severe Case Occurring in a Boston Physician

BY JOHN F. CASEY, M.D.*

A RECENT letter to the doctors of Massachusetts from Dr. Henry D. Chadwick, Commissioner of Public Health,[1] calls attention to the increase of undulant fever in Massachusetts. During 1935, 42 cases were reported in the State, 1 of which was fatal.

For about ten years, this disease has been present in the western and southern parts of this country; but New England has been comparatively free from it. The disease has been reportable in Massachusetts only since 1930.

In 1930, 6 cases were reported; in 1931, 15 cases, in 1932, 15 cases; in 1933, 11 cases; and in 1934, 15 cases.[2] The increase of reported cases from 15 in 1934 to 42 in 1935 is probably due to an increase in the incidence of the disease, for the annual report of the Massachusetts Department of Public Health for the year ending November 30, 1934 states that the incidence of contagious abortion among the dairy cattle of the State was very high. It is possible, however, that there are many undiagnosed cases in the State, for the symptoms are many and varied.

Having cared for a medical colleague through a severe infection with *Brucella abortus*, lasting from the end of May to September, it seems important to call the attention of the New England physicians to the various aspects of this disease which may cause such serious illness and great economic loss.

For years, this disease has been known by various names,—Malta fever, Mediterranean fever, and undulant fever are a few. Recently, both in this country and abroad, the name *brucellosis* has been adopted. The disease was first limited to the Island of Malta and neighboring islands; but has gradually spread throughout Europe and America.

Historical: In 1887, David Bruce,[3] visiting Malta on his honeymoon, became so much interested in the disease that, calling on his wife to help him, he attacked the bacteriologic problem. From the spleens of soldiers, who had died of the disease, he grew a micrococcus, the *Micrococcus melitensis*. Mosquitoes, flies, fleas, the water supply and foodstuffs were investigated. Attention was finally focused on the goats which supply most of the milk on the Island of Malta. Bruce showed that 50 per cent of the goats were infected and proved that the disease was transmitted through their milk. From this medical pioneering of the Colonel and his lady, we get the name, *brucellosis*.

About ten years after Bruce had discovered the *Micrococcus melitensis*, a Danish veterinarian, Bang, discovered the organism causing contagious abortion in cows, and named it *Bacillus abortus*.

In 1917 Alice Evans, a young bacteriologist in the United States Department of Agriculture, discovered the close relationship between the *Micrococcus melitensis* of Malta fever and the *Bacillus abortus* found in the milk of American herds. Later the generic name of *brucella* was given to the group.

In 1924 Keefer[4] reported the first human brucellosis case in this country. Since that time the disease has been found in every part of this country, but particularly in the great cattle raising states.

Clinical Types of the Disease: There are acute, mild and severe, types; and also a chronic type. The acute type may become chronic. In various parts of the country, there are patients who have been ill for over a year with the disease.

Incubation Period: From all available data, it appears to be two weeks, although there is some evidence that the disease may occur as early as one week after exposure.

Symptoms: In acute brucellosis, the usual symptoms are fever, chills, drenching sweats, severe constipation, abdominal discomfort, arthritic and neuritic pains, nausea, occasional vomiting, and loss of appetite with resulting great loss of weight and strength. A mousey odor may be detected about the patient but this is more often noticed by the patient himself.

In the chronic form, exhaustion, insomnia, irritability, and aches and pains for which no objective causes can be found form the common picture.

Physical Signs: The physical signs offer little aid in diagnosis. There are often no signs except those due to fever and anemia. When the disease has been present for some time, emaciation may be marked. The spleen is frequently enlarged, occasionally the liver.

Diagnosis: In any continued fever without localizing signs, think of brucellosis, and have a blood examination. Typhoid fever, tuberculosis, rheumatism, influenza, subacute bacterial endocarditis, malaria, and neurasthenia are some of

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the diagnoses erroneously made. Sidel and Segal[5] report two cases in *The New England Journal of Medicine* in which the diagnosis at first in one case was subacute bacterial endocarditis, in the other pulmonary tuberculosis.

Subacute bacterial endocarditis, however, may be due to the brucella organism. Angle[6] has reported 100 cases of brucellosis with two deaths, both from a complicating subacute bacterial endocarditis in which brucella was shown to be the etiologic organism.

It is important to know that brucella may be the cause of meningitis. Sanders[7] in 1931 reported a fatal case with recovery of the organism from the spinal fluid. Poston and Smith[8] have recently reported two cases that recovered, and have reviewed the literature.

In brucella meningitis, the usual signs of meningitis are present, the spinal fluid is under pressure, is turbid, and shows a cell count of 500 to 1,000, mostly polymorphonuclear leucocytes. The brucella may be cultured from the spinal fluid by a special method used by Poston and Smith.

Hartley and co-workers[9] report a case in which they were unable to grow the organism from the spinal fluid but recovered it after guinea-pig inoculation.

In a recent article Alice Evans[10] calls attention to obscure types of brucellosis. The literature preceding this article contains little about the chronic form of the disease. It is not surprising that these cases that rarely run a noticeable temperature over months of illness and that complain of exhaustion, insomnia, irritability and indefinite aches and pains for which no localizing signs are found, are often diagnosed as neurasthenia.

Hardy, Jordan and Borts,[11] in a paper read at the last American Medical Association meeting, present a complete study on 705 cases occurring in the State of Iowa in the last six years. Their study reveals that the organism has been the cause of many and varied medical and surgical conditions, such as chronic abscesses, osteomyelitis, pleural effusion, endocarditis, pericarditis, arthritis, spondylitis and meningitis.

Laboratory Aids in Diagnosis: The agglutination test is of great value; but too much reliance must not be placed upon it. Blood is taken as for a Wassermann test and sent to the State Department of Public Health Laboratory. Many men have felt that when the test is reported positive in dilutions of 1 to 40, or 1 to 50, it was not significant, as, in these strengths, agglutination, at times, takes place with the serums of patients suffering from other fevers. One to 80 or higher dilutions were regarded as necessary for a diagnosis.

Even this is misleading, however, for it is now known that brucellosis may be the correct diagnosis even if the agglutination test is positive only in low dilution or, at times, when the agglutination test is negative.

Harris[12] reports 75 cases of chronic brucellosis that he has observed. Eleven of these had repeated negative agglutination tests although the other evidence proved them undoubted brucellosis cases.

The skin test has proved very useful. Two to four hundredths cc. of brucella antigen (any of the brucella vaccines on the market may be used) is injected intradermally. At the same time, a normal person is injected. The antigen usually produces a tuberculin-like reaction in the patient within eight hours that persists for twenty-four to forty-eight hours, whereas little or no reaction is observed in the control.

Blood cultures are not of value for quick diagnosis. Special media must be used. The organism is slow to grow. Cultures should not be reported negative until they have been observed for thirty days. There are many different strains of the brucella organism and special media and bacteriologic technic are needed to grow and identify the various strains.

The blood shows a secondary anemia, and a leucopenia is usually found by the time the patient is seen although there is an early leucocytosis.

CASE REPORT: A Boston specialist of 40 had a slight chill following a golf game on May 17, 1935. He felt well that night and the following day continued at work; but, in the afternoon, felt flushed and was found to have a temperature of 100.4°. From that time until August 2, he had a fever some part of each day.

Past History: He had always enjoyed good health except for a severe influenza pneumonia in 1918.

Physical Signs: At the onset of the disease, he was an exceptionally healthy and well-developed individual. He was normal in every particular except for a moderately coated tongue. Later in the disease, the only signs he showed were those due to fever and loss of weight. At no time was any enlargement of the spleen or liver demonstrated.

Progress of the Disease: During the first few days, he felt perfectly well most of the time and was up and about. But in the afternoons he felt chilly, and then his temperature rose to 100° or 100.6°, and was followed during the evening by a moderate sweat. When this had occurred for three successive days and no focus of disease was apparent, he was asked to report to the hospital for complete study.

X-ray of the teeth showed nothing abnormal. Heart x-ray was normal. X-ray of the lungs disclosed a moderate shadow at the right base. This was the site of his old pneumonia. It was important to know this, for, at first, it was thought that this might be a new process and the cause of his present illness.

Laboratory work showed the following: Hg. 83 per cent; R.B.C. 4,320,000; W.B.C. 16,800; Polys. 78 per cent; Blood culture, sterile. Blood smears showed no malaria parasites. Blood was sent to the State Laboratory for the agglutination test for undulant fever, and to the United States Public Health Laboratory for tests for undulant fever and tularemia. The tularemia test was negative; but the

serum gave complete agglutination of *Bacillus abortus* in dilution of 1:640 and later, on June 1, 1:1280.

We made several attempts to grow the organism from the blood, but were unsuccessful.

The white blood counts were interesting, showing the early leucocytosis with rapidly developing leucopenia, and the increase in leucocytes due to foreign protein.

May 26	16,800	
May 28	13,600	
May 29	10,800	
June 2	7,200	
June 3	6,700	
June 5 a. m.	6,000	Before serum.
June 5 p. m.	7,500	Serum given June 5, 6, 7.
June 6	10,600	
June 9	10,250	
June 10	12,600	
July 11	6,400	After relapse.
July 17	11,800	Daily injections of liver given since July 11.

Urine: Several examinations, sp. gr. 1.012; alb. 0; sugar 0; sediment, negative.

A skin test was done with killed brucella organisms and was positive; one done on a control was negative.

The patient left the hospital on June 11 and went to the beach. He remained in bed most of the time; but spent part of the day out-of-doors walking about. Each day he felt well, but at night he had a slight fever and a moderate sweat. On July 4, he had a severe chill followed by a temperature of 103° and later, a drenching sweat. He was then more severely ill than at any time in the disease. He had continual nausea, vomiting at times, no appetite, great abdominal discomfort, severe constipation, and a neuralgic pain in back of one eye. He continued severely ill until July 14 when his symptoms began to abate. During this period he had one sweat lasting 5 hours. He had lost weight rapidly, at the low point of his illness having lost 50 pounds. He improved gradually, having a fever some part of every day until August 2 when the fever ceased abruptly. From that time on, he improved rapidly. He returned to work on September 3, still about 30 pounds under his usual weight. By January, 1936, he had regained his lost weight and has been in excellent health since.

Treatment: As in all diseases in which no proved specific has been discovered, the number of remedies are multiple. In our case, we had two medications which we thought of value. From the start of the disease, the patient took from 40 to 60 grains of acetylsalicylic acid a day. We felt that this added to his comfort, and when the amount of the medication was cut down he began to suffer from joint and muscle pains. At times, the medication was omitted because the patient felt that it caused his sweats to be more profuse.

Hoping to prevent the usual anemia present in the disease, we gave frequent intramuscular injections of liver. It seemed to be of value. The low point of the blood was a hemoglobin of 74 per cent and a red blood cell count of 3,760,000.

We had many advisers in regard to specific therapy, and elected to use an antiserum prepared by Foshay[13] and his associates of the University of Cincinnati. This serum was obtained from goats after subcutaneous inoculations with chemically treated suspensions of brucellae. In view of the fact that the last dose of serum was given on June 7 and the fever did not terminate until August 2, we feel that either our dosage was not sufficient or that the serum was without value. We are inclined to think that the dosage was insufficient, since the patient showed a decided improvement following the use of the serum, but suffered a relapse on July 4.

Bannick and Magath[14] of the Mayo Clinic recently reported two cases treated with the Foshay serum. They feel that the serum was of definite value in these cases. At the time of treating our case the serum was obtained from goats. Foshay now uses horses instead and considers the serum more potent.

Many men who have treated a great number of cases, both in this country and abroad, feel that a brucella vaccine is of great value. Several commercial houses have a vaccine on the market prepared according to the method of Simpson. It is widely used. Angle, who has developed his own vaccine, speaking of vaccine therapy says, "I am convinced that it is the best single therapeutic agent now available." The vaccine causes a severe reaction. This is both a pronounced local and general reaction. It is so severe that the use of vaccine is contraindicated in elderly individuals.

The latest form of treatment reported is the production of hyperpyrexia by means of the Simpson-Kettering hypertherm. Prickman and Popp[15] of the Mayo Clinic report three cases treated in this manner with good results. In their opinion the results justify a further trial of this method.

Because of the varied length of illness in brucellosis and because of the frequent abrupt termination of the illness in many cases, no matter what form of therapy is used, it is difficult to evaluate the various forms of treatment.

Source of Infection in Our Case. The question most often asked about our case was, "Where did he get the disease?" The patient had not been outside of Massachusetts for several months. There were no known cases in Boston or vicinity. He used only pasteurized milk and cream in his home, and it was unusual for him to drink milk. In spite of careful investigation by the State Department of Public Health, the source of his infection was not determined.

Comment: The disease brucellosis is a severe disease of long duration. It may involve almost any part of the body, in this respect resembling tuberculosis and syphilis. Pasteurization of milk would eliminate the source of most of the brucellosis in this country. We of Massachu-

setts are fortunate in that 85 per cent of the population of our state is protected by the use of pasteurized milk.

It might be well, however, to call attention to the fact that some areas are not protected, to mention one, our island of Nantucket, where raw milk is commonly used.

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MILK MODIFICATION AND INFANT CONSTITUTION*

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MILK constitutes the basis of formulas for infants' feedings. Proper choice of the milk suited to the particular infant is indispensable in successful infant nutrition. The great variety of milks available make such a choice difficult in the individual infant because of unnecessary empiricism in practice. More stress has been placed upon the various milks and their properties than on infants and their tolerance. Nutritional knowledge has advanced sufficiently to adapt effectively the required type of milk to the individual infant rather than the infant to the milk.

The type of milk selected requires modification, the procedure of choice depending upon the infant. The resulting formula may meet all the nutritional needs of the infant and yet be unsuited to his digestive tolerance. The theoretical goal in nutrition is the same for all infants but the methods of attaining it necessarily differ with each infant. Goal and method are not only necessary but inseparable in effective infant feeding. Thus, there can be no single formula for the universal feeding of infants. The percentage composition of cow's milk mixtures, imitating breast milk, becomes less important than the behavior in the infant's stomach. We have studied the response of various methods of feeding in terms of the infant's constitution as a more physiologic basis for determining the type of formula indicated.

PLAN OF INFANT FEEDING STUDY

Comparative studies of "standard formulas" prepared from various types of milk were made on infants during the first three months of life. The newborns on the service were maintained from birth with the hydrating solution given at two-hour intervals throughout the twenty-four hour cycle and put on the formula under study on the second day of life. An effort was made in each case to have the infant nurse and the breast-fed infants were used as controls. Despite the willingness of most mothers to nurse their babies and our insistence in favor of breast feeding, fewer infants were nursed than is considered good practice. Although theoretically tuberculosis in the mother is the only contradiction to nursing, actually we have found severe postpartum disturbances affecting the mother physically and temperamentally to an extent that made nursing very trying. Breast pumps were tolerated for short periods and manual stripping was rejected by many mothers. Whatever the maternal condition, the baby was weaned if the quantity of breast milk was less than an average of two ounces per feeding after the first fortnight.

Weaning during the newborn period was instituted for very definite indications and not for the purpose of these studies. Many of the infants, included in the data, nursed for a number of days during which time the intake of the special formula was relatively small. But during the baby's stay in the nursery the intake of the milk mixture was gradually increased until the formula under study comprised the total day's feeding. The infants' progress was also under the supervision of the research dietitian, Miss McMahon and the technical data under Miss Pugsley, research chemist, both devoting full time to these nutritional studies.

Over fifty well newborns were observed serially on each formula of fresh, powdered, evap-

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orated and acid milks* with breast milk as the control. Careful records were made of the physical status and functional response to the particular feeding. This was continued at home after the babies were discharged from the nursery and observed subsequently at weekly intervals in a well-baby unit where clinical and laboratory examinations were made. Those infants whose mothers failed to keep appointments were followed up at home by one of the dietitians. The mother was provided with the special milk used for the infant's formula thus insuring constancy of product and of cooperation. Only normal, well infants were included in this series. It is the first three months of the feeding progress of these infants that concerns this report.

ROUTINE FEEDING OF "STANDARD" FORMULAS IN WELL INFANTS

I. Control Series. Fifty-five normal infants were maintained exclusively on breast milk for three months. The breast feeding was started five hours after birth in newborns with a birth-weight over six pounds. Although five of these newborns had difficult deliveries they showed no postnatal manifestations. Orange juice and cod liver oil were introduced the third week. The orange juice was first given half strength and gradually increased so that two ounces were given by the end of the first month. The 10 D cod liver oil* was given five drops a day increasing it gradually until each infant received one teaspoonful a day. The average volume intake was 3.8 ounces per feeding during the first month, subsequent amounts not being determined. Indeed it was difficult to obtain a sufficient number of weighings during the first month to estimate the average volume intake, the accuracy is therefore indeterminate for comparison with the equivalent values for artificial feedings. The average volume intake consumed during the twenty-four hour cycle was, accordingly, 22 ounces. The average calorie intake was about 56 calories per pound per day. The average gain in weight was 6.9 ounces per week and the average gain in height 1 inch per month. The average number of stools was 2.5 per day. Three of the infants developed transient upper respiratory infections; four had alimentary disturbances, that is, one vomited, one had colic and two developed diarrhea.

Series II. *Whole Milk Formula.* Fifty-four normal infants were maintained on a whole milk mixture for three months. It consisted of boiled whole milk with 10 per cent added carbohydrate. The formula was started the second day of life in well newborns with a birth weight over six pounds. Although seven of the group studied had difficult deliveries there were no postnatal manifestations. Orange juice and cod liver oil were introduced the third week. The orange juice was first given half strength and increased gradually so that two ounces were given by the end of the first month. The 10 D cod liver oil was offered five drops a day increasing it gradually until the infant received one teaspoonful a day. The formula was made up for twenty-four hours and divided into six feedings given at four-hour intervals during the first month and subsequently into five feedings. The amount offered at each feeding was slightly above that usually taken by infants at each age level.

The average volume consumed by an infant was 4.0 ounces per feeding during the first month, 5.2 during the second and 6.0 during the third month, the average volume for the total day's feeding being 24, 26 and 30 ounces respectively. The average calorie intake was about 60 calories per pound per day. The average gain in weight was 6.0 ounces per week and the gain in height was 1 inch per month. The average number of stools was 3.3 per day. There were three cases of upper respiratory infection, one furunculosis and one pyelitis. But thirteen infants showed other disturbances, that is, three vomited, three had colic, one had diarrhea, four were severely constipated, one developed eczema and one had pyloric stenosis.

The distribution of alimentary upsets was not uniform at each age level, the greatest proportion being during the first month and the least during the third month: 36 per cent during the first month, 24 per cent during the second and 11 per cent during the third month with an average of 24 per cent for the entire period. Several infants with limited digestive capacities were eliminated from this study during the first month. The most striking feature was the correlation between alimentary tolerance and body build. About 80 per cent who showed digestive disturbances were of the lateral type. Apparently the majority of normal infants of linear body build are able to take undiluted boiled whole milk in amounts adequate to satisfy the appetite without revealing intestinal disturbances.

Series III. *Diluted Milk Formula.* Fifty-nine normal infants were maintained on a diluted milk formula for three months. It consisted of boiled half-strength milk with 10 per cent added carbohydrate. The formula was started the second day of life in well newborns with a birth weight over 6 pounds. Although three of the group studied had difficult deliveries there were no postnatal manifestations. Orange juice and cod liver oil were introduced the third week. The orange juice was first given half strength and increased gradually so that 2 ounces were given by the end of the first month. The 10 D cod liver oil was offered five drops a day increasing it gradually until the infant re-

*Supplied through the courtesy of Mead, Johnson & Company.

ceived one teaspoonful a day. The formula was made up for twenty-four hours and divided into six feedings given at four-hour intervals during the first month and subsequently into five feedings. The amount offered at each feeding was slightly above that usually taken by infants at each age level.

The average volume consumed per infant was 4.2 ounces per feeding during the first month, 5.4 during the second and 6.3 during the third month, the average volume for the total day's feeding being 25, 27 and 32 ounces respectively. The average caloric intake was about 52 calories per pound per day. The average gain in weight was 5.9 ounces per week and the average gain in height was one inch per month. The average number of stools was 2.5 per day. There were three cases of upper respiratory infection, six cases of alimentary disturbances, i.e., three vomited, two developed colic and one had diarrhea. The distribution of alimentary upsets was not uniform but in the opposite sense of the group on whole milk formulas. There appeared a somewhat greater number of disturbances during the second and third months than during the first. Apparently dilution does not spare infants intestinal disturbances after the first month.

Series IV. *Evaporated Milk Formula.* Fifty-eight normal infants were maintained on an evaporated milk formula for three months. It consisted of evaporated milk, 1 to 2 dilution, with 10 per cent added carbohydrate. The formula was started the second day of life in well newborns with a birth weight over 6 pounds. Although six of the group studied had difficult deliveries there were no postnatal manifestations. Orange juice and cod liver oil were introduced the third week. The orange juice was first given half strength and increased gradually so that 2 ounces were given by the end of the first month. The 10 D cod liver oil was offered 5 drops a day increasing it gradually until the infant received 1 teaspoonful a day. The formula was made up for twenty-four hours and divided into 6 feedings given at four-hour intervals during the first month and subsequently into five feedings. The amount offered at each feeding was slightly above that usually taken by infants at each age level.

The average volume consumed per infant was 4.5 ounces per feeding during the first month, 5.0 during the second and 6.3 during the third month, the average volume for the total day's feeding being 25, 25 and 30 ounces respectively. The average caloric intake was about 66 calories per pound per day. The average gain in weight was 7.1 ounces per week and the average gain in height was one inch per month. The average number of stools was 2.6 per day. There were four cases of upper respiratory infection. Five developed alimentary disturbances, that is, two vomited, one had colic, one diarrhea and one vomited associated with asthma.

Series V. *Powdered Milk Formula.* Sixty normal infants were maintained on a powdered milk formula for three months. It consisted of powdered milk, 1 tablespoonful to 2 ounces of water, with 10 per cent added carbohydrate. The formula was started the second day of life in well newborns with a birth weight over 6 pounds. Although five of the group studied had difficult deliveries there were no postnatal manifestations. Orange juice and cod liver oil were introduced the third week. The orange juice was first given half strength and increased gradually so that 2 ounces were given by the end of the first month. The 10 D cod liver oil was offered 5 drops a day increasing it gradually until the infant received 1 teaspoonful a day. The formula was made up for twenty-four hours and divided into six feedings given at four-hour intervals during the first month and subsequently into five feedings. The amount offered at each feeding was slightly above that usually taken by infants at each age level.

The average volume consumed per infant was 4.1 ounces per feeding during the first month, 5.1 during the second and 5.9 during the third month, the average volume for the total day's feeding being 24, 26, and 29 ounces respectively. The average caloric intake was about 60 calories per pound per day. The average gain in weight was 6.4 ounces per week and the average gain in height was one inch per month. The average number of stools was 2.4 per day. There were five cases of upper respiratory infection including one with otitis media. Five developed alimentary disturbances, that is, two vomited, one had colic, three developed diarrhea, one of which was secondary to an upper respiratory infection.

Series VI. *Acid Milk Formula.* Fifty-six normal infants were maintained on powdered lactic acid milk formula for three months. It consisted of 1 tablespoonful of the powder to 2 ounces of water, with 10 per cent added carbohydrate. The formula was started the second day of life in well newborns with a birth weight over 6 pounds. Although seven of the group studied had difficult deliveries there were no postnatal manifestations. Orange juice and cod liver oil were introduced the third week. The orange juice was first given half strength and increased gradually so that 2 ounces were given by the end of the first month. The 10 D cod liver oil was offered 5 drops a day increasing it gradually until the infant received 1 teaspoonful a day. The formula was made up for twenty-four hours and divided into six feedings given at four-hour intervals during the first month and subsequently into five feedings.

The amount offered at each feeding was slightly above that usually taken by infants at each age level.

The average volume consumed by each infant was 3.8 ounces per feeding during the first month, 5.3 during the second and 6.2 during the third month, the average volume for the total day's feeding being 22, 26, and 31 ounces per pound per day. The acid feedings were taken with more avidity after the first month. The average calorie intake was about 67 calories per pound per day. The average gain in weight was 7.1 ounces per week and the average gain in height was one inch per month. The average number of stools was 2.7 per day. There were three cases of upper respiratory infection. Five infants developed alimentary disturbances, that is, one vomited, two had colic, two diarrhea and the feedings were reduced.

INDIVIDUAL FEEDING OF "STANDARD" FORMULAS IN WELL INFANTS

Every type of formula tested was effective routinely in over 80 per cent of the infants studied. But in each group there were always a number of nonconformists. The particular formula was not adapted to them and they reacted with alimentary offense. Even mother's milk yielded its quota of digestive intolerance. The difficulty is with the infant in each group and not with any of the formulas tested. It appears improbable that any one type of feeding will ever be universally applicable to all infants.

The five series of routine formula feedings separated the normal infants from those with latent constitutional disorders. And even among the large group of normal infants in each series there were differences in the relative tolerance for milk as the first "solid" food. The range of digestive tolerance is so wide in most infants that no abnormal responses are manifest clinically with modified milk mixtures. But in infants with latent constitutional disorders—developmental, allergic, neuropathic—there is either a low digestive capacity for any food or a diminished tolerance for cow's milk, frequently below maintenance requirement. Not all these infants rebel against "standard" formulas because the infant's functional deviations may be slow in becoming manifest.

But the constitutional stigmata persist although not always poignantly. The routine feedings unsuited to these infants may tide them over the first months of difficult adjustment but subsequently contribute to the precipitation of their constitutional disease. What may develop will appear more violently if irritated by inappropriate feedings from birth. The feeding of routine formulas to well infants from birth ignores the prevalence of latent constitutional disturbances. Many of these can be recognized early and an appropriate formula adapted to their more limited digestive capacities.

We attempted to distinguish normal from diathetic newborns to determine the type of milk indicated for each infant individually. Infants with overt disturbances were excluded from this study. Of the forty-five infants with diatheses, 65 per cent were allergic, 30 per cent neuropathic and 5 per cent developmental in origin. The infants that developed allergic manifestations showed enough suggestion of latent allergy to warrant early adaptation of the formula. The most helpful sign was an erythematous linear fold in the postauricular region or beneath the lobe of the ear, more striking beneath the left than the right ear. In addition the usual premonitory expressions of allergy such as a hairless scaly scalp and desquamated dry skin were helpful. When these manifestations in the newborn were corroborated by an allergic history in either parent, the infant was considered a problem in latent allergy. Neuropathic infants were recognized by hypertonic manifestations during the first days of life. There was predominance of the flexor muscles with generalized and continued spasticity, restlessness, sleeplessness and irritability. Infants with latent developmental disorders were too varied to permit of any single procedure to be of value for early diagnosis.

Series VII. *Individualized Formulas.* Fifty normal infants were maintained on individualized formulas for three months. The type of feeding selected was in accordance with the indications of the individual infant. Long lean infants were given 75 calories per pound of body weight per day and lateral infants were given 60 calories. The former were fed concentrated whole milk mixtures, three to one dilution with 10 per cent added carbohydrate and the latter diluted milk mixtures, one to one dilution with the same amount of added carbohydrate. Infants with latent allergy were given evaporated milk, one to two dilution with 10 per cent added carbohydrate, with a caloric intake depending upon their body build. Infants with hypertonia were maintained on acid milk, 1 tablespoonful to 2 ounces of water with 10 per cent added carbohydrate.

The formulas were started the second day of life in well newborns with a birth weight over 6 pounds. Although four of the group studied had difficult deliveries there were no postnatal manifestations. Orange juice and cod liver oil were introduced the third week. The orange juice was first given half strength and increased gradually so that 2 ounces were given by the end of the first month. The 10 D cod liver oil was offered 5 drops a day increasing it gradually until the infant received 1 teaspoonful a day. The formulas were made up for

twenty-four hours and divided into six feedings given at four-hour intervals during the first month and subsequently into five feedings. The amount offered at each feeding was slightly above that usually taken by infants at each age level.

The average volume consumed per infant was 4.4 ounces per feeding during the first month, 5.5 during the second and 6.5 during the third month, the average volume for the total day's feedings being 26, 27 and 30 ounces respectively. The average caloric intake was about 70 calories per pound per day. The average gain in weight was 7.3 ounces per week and the average gain in height one inch per month. The average number of stools was 2.6 per day. There were four cases of mild upper respiratory infection and one infant vomited.

EVALUATION OF RESULTS WITH ROUTINE AND INDIVIDUALIZED FEEDING

I. *Comparative Intake of Formulas.* The ingestion of various types of formulas was relatively uniform in amount for each group studied. During the first month the infants on breast milk took such varied amounts at each feeding that the average volume intake is of little significance in comparison with artificial feeding. Apparently the effort exerted in nursing is more of a factor than appetite in determining the volume intake at a feeding. The placid role of infants on artificial feeding accounts for the relatively uniform intake on the various types of prescribed formulas.

Infants took less of the acid formula during the first month but, once they became adapted to it, they took more of that formula than of the others. During the entire period of study, infants took more of processed milk formulas than of the whole milk formula. And those on individualized feedings consumed more per feeding as well as for the twenty-four hour cycle. This is partly due to the fact that more infants were included in the series who have taken feedings steadily without alimentary upsets. The caloric intake per pound of body weight per twenty-four hours was greater on the more concentrated feedings, the least being with the diluted milk mixtures and the most with the acid and individualized formulas.

II. *Comparative Tolerance of Formulas.* Food tolerance is estimated clinically without quantitative criteria. It is difficult to make comparative estimates of the relative degrees of tolerance for the formulas tested. Quantitatively an infant's tolerance for a food represents the ratio of the maximum amount of the food he can metabolize without resultant disturbances by the maximum amount of that food metabolized by a normal infant of the same age group. We must necessarily deal with maximum rather than with optimum intake in the consideration of tolerance because we do not know what the optimum of any food is quantitatively for any infant. But we do know that there is a wide range between the minimum and the maximum and that any attempt to exceed the maximum produced a variety of disturbances essentially alimentary in nature. We are, therefore, led to the evaluation of tolerance of various formulas by a negative qualitative method of procedure. Those formulas consumed in the required amounts were considered well tolerated if they produced no untoward symptoms while those which produced digestive disturbances apparently exceeded the digestive capacities of the infants and were considered poorly tolerated.

All types of formulas were well tolerated, excepting the whole milk mixture during the first month. There were the least number of digestive upsets on the individualized formulas and on the breast feeding throughout the feeding period studied. There were fewer digestive disturbances on breast feeding than on fresh milk formulas. Only significant symptoms of digestive disturbances were included in this evaluation of tolerance for much of the minor symptomatology was more an expression of mismanagement than of misfeeding. However, a feature that is frequently neglected in relation to food tolerance during the first three months of life is the increased nerve tone of infants in both breast and bottle feeding. It is usually alleviated by transient modification of the feeding quantitatively or qualitatively rather than by resorting to sedatives. The number and quality of the stools were not considered diagnostic of the normal range of alimentary function, although frequency or abnormal character of stools was deemed pathognomonic of gastrointestinal disturbance.

A striking feature of the two groups on the fresh milk mixtures was the relation between milk tolerance and body build in well infants. During the first two months there was a relatively great preponderance of digestive disturbances among those infants who were maintained on the whole milk formulas. Eighty per cent of those infants were of the lateral body build. Most of the linear type infants tolerated whole milk mixtures without any disturbances. Apart from food tolerance the caloric requirement from body surface considerations indicates that the linear infants require about 70 calories per pound of body weight while the lateral infants require about 55 calories. This difference in energy requirement appears to parallel their difference in tolerance. It is curious that the high gastric acidity during the first week of life should not increase the digestive capacities of all infants sufficiently to enable them to tolerate whole milk formulas. But

after the second month the percentage of disturbances was parallel to those in the other groups.

A significant number of digestive upsets occurred in the group maintained on breast feeding. Excluding disturbances resulting from faulty technic in nursing, those that persisted were of the same order of magnitude as in the infants on artificial feeding. The time-honored procedure of analyzing breast milk chemically for the gross constituents is considered irrelevant. Even analysis of the minutiae is unnecessary because deficiency disease has a long latent period before inducing disturbances. The difficulty, we believe, is usually with the infant constitutionally and not with the milk chemically. But weaning was not deemed necessary until supplementary procedures failed to allay the disturbance.

The formulas prepared from processed milk were the best tolerated. Offhand, these would appear the most logical for routine use in infant feeding if nursing is impossible, but such an approach defeats the very purpose of infant nutrition. The type of feeding that meets with the digestive capacities of the infants is indicated rather than that which least utilizes digestive mechanisms. It is the newborn's task to adapt to "foreign" foods—a biologic and therefore a physical necessity. Ability to digest a food is gained by ingesting the food in gradually increasing amounts. The more work the digestive mechanism is made to perform, the greater the alimentary efficiency. The indications in normal infant feeding are fresh milk mixtures for the infants that can tolerate them and processed milk mixtures for those with diminished digestive capacities.

III. *Comparative Nutritional Status.* The adequacy of each type of feeding is best determined by the character and rate of growth of the infants of each group. But again growth evaluation is clinical rather than quantitative because we have no absolute standards of the composites of growth for each age level. Average weights and measurements of infants cannot be taken as guides for the developmental progress of an individual infant, for the average infant is nonexistent. Developmental progress of an infant can best be evaluated in terms of himself as a standard and the increments of growth occurring at periodic intervals as the individual criteria of satisfactory achievement. These are based more upon clinical judgment than upon mere measurement. They include a variety of external manifestations of well-being such as type of facies, color of mucous membranes, condition of skin, quality of subcutaneous tissues, tissue turgor, muscle tone and skeletal characteristics. Even these properties of an infant are, during the first weeks of life, more an expression of his heredity and constitution than of the type of feeding instituted.

Each group of infants thrived on the various formulas as on the breast feeding. The greatest gain in weight was in those on individualized feeding and the least on the diluted milk formulas. The infants on breast feeding and on the processed milk formulas gained more rapidly than those on the fresh milk mixtures. The more concentrated the formulas the more rapid is the weekly gain in weight. In no groups did this necessarily represent mere accumulation of adipose tissue. Despite the high gains in weight there were no manifestations of overfeeding. In fact we have observed no absolute relation between the high gains in weight and quantities of formulas ingested. This applies to both the breast and artificially fed infants. Large lateral infants, for example, with birth weights of about 8 pounds made more satisfactory gains on 20 ounces of a formula per twenty-four hours than did the long lean infants with a birth weight of about 7 pounds on an intake of 25 ounces. But on all types of feedings the gain in height was practically the same at least for the first three months. In each group the incidence of infection was about the same, acquired in most infants from adult contacts.

CONCLUSIONS

1. Comparative studies of "standard" formulas were made on 337 infants during the first three months of life with fifty-five breast fed infants as controls. The formulas consisted of whole milk, half diluted milk, evaporated milk (1:1), powdered whole milk (1 tablespoonful to 2 ounces of water), powdered lactic acid milk (1 tablespoonful to 2 ounces of water), each reinforced with 10 per cent added carbohydrate. Each group maintained on a routine formula was compared with a group of fifty infants given individualized formulas.

2. The infants consumed more of the processed milk formulas than of the fresh milk formulas and the maximum was on individualized feedings throughout the period of study. The least number of digestive disturbances was observed on breast feeding and on individualized formulas.

3. The linear type infant required about 70 calories per pound of body weight and tolerated concentrated milk mixtures while the lateral type infant required about 55 calories per pound of body weight and tolerated diluted milk mixtures better.

4. Maximum gains in weight were observed in infants on individualized feeding and the minimum on diluted milk formulas. Infants on breast feedings and on processed milk feedings

gained more rapidly than those on the fresh milk mixtures. The gains in height were the same on all types of feeding during the first three months. The incidence of infection was about the same for each group of infants studied.

5. Standard routine formulas were effective in over 80 per cent of the infants studied. The residual nonconformists represented infants with latent constitutional disorders—organic, allergic and neuropathic—with either lowered digestive capacities or diminished tolerance. The early recognition of these types displaced routine by individualized feeding. This group showed the highest percentage of successful infant feeding.

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COMPARATIVE RESULTS ON ROUTINE AND INDIVIDUAL FORMULAS

	Breast Milk	Whole Milk Formula	Diluted Milk Formula	Evaporated Milk Formula	Powdered Milk Formula	Acid Milk Formula	Individualized Formula
Age of Infants (Wks.)	1-12	1-12	1-12	1-12	1-12	1-12	1-12
No. Cases	55	54	59	58	60	56	50
Difficult Deliveries	5	7	3	6	5	7	4
Duration of Feeding (Days)	85	80	82	86	85	88	85
Supplements	OJ&CLO*	OJ&CLO	OJ&CLO	OJ&CLO	OJ&CLO	OJ&CLO	OJ&CLO
Av. Vol./Feedings (Ozs.)							
1 mo.	(3.8)	4.0	4.2	4.2	4.1	3.8	4.4
2 mo.	—	5.2	5.4	5.0	5.1	5.3	5.5
3 mo.	—	6.0	6.5	6.1	5.9	6.2	6.5
Av. Vol./Day Feedings (Ozs.)							
1 mo.	(22)	24	25	25	24	22	26
2 mo.	—	26	27	25	26	26	27
3 mo.	—	30	32	31	29	31	33
Av. Intake Clas./Lb./Day	(56)	60	52	66	60	67	70
Av. Gain Wt. Ozs./Wk.	6.9	6.0	5.9	7.1	6.4	7.1	7.3
Av. Gain Ht. (Mo.)	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Av. No. Stools/Day	2.5	3.3	2.5	2.6	2.4	2.7	2.6
% Digestive Disturbances	8	24	12	9	11	10	2
% Infections	7	9	5	8	10	6	8
% Successful Feeding	90	70	85	88	86	88	95

*OJ = Orange juice.
CLO = Cod liver oil.

MILKS INDICATED FOR INFANTS

INFANT TYPES
(392 Babies)

Linear (20%)	Medial (25%)	Lateral (55%)
Breast Milk	Breast Milk	Breast Milk
Cow's Milk (3:1)	Cow's Milk (2:1)	Cow's Milk (1:1)
Evaporated Milk (1:1)	Evaporated Milk (1:1½)	Evaporated Milk (1:2)
Powdered Milk (1:7)	Powdered Milk (1:8)	Powdered Milk (1:10)

INFANT DIATHESES
(45 Cases)

Allergic (65%)	Neuropathic (30%)	Developmental (5%)
Breast Milk	Breast Milk	Breast Milk
Goat's Milk	Thick Feeding	Evaporated Milk
Evaporated Milk	Evaporated Milk	Acid Milk
Powdered Milk	Acid Milk	
Vegetable Milk		

VERMONT STATE MEDICAL SOCIETY

MEDICAL SERVICE IN VERMONT: SOME CHANGES IN FORTY YEARS*

BY L. W. BURBANK, M.D.†

I WISH to call your attention to some of the changes which have taken place in medical service in rural sections of Vermont during the past forty years. I refer especially to methods of dealing with those diseases which are communicable.

The centralization of medical practice has increased more in twenty-five years than in a full century before that time. Since 1896, sixteen years have been added to the average span of human life. That is a larger increase than has taken place in any other forty years since the beginning of recorded history. In the rural sections of Vermont, the chief gain along this line was due to the efforts of the family physician. He did most of the preventive work until about 1920. Since that time the work has been done more and more by social workers from the larger towns.

The town of Cabot, thirty-six square miles in area, has been for forty years the home of about eleven hundred persons, nearly all farmers. Cabot village, about 1150 feet above sea level, has a population of about 225.

In the northern corner of the town rises the Winooski River which flows to the southwest, down a narrow valley through Marshfield, a smaller town with a population of 900, and Plainfield. This valley is bounded on the northwest by the mountains of Woodbury and Calais; on the north by those of Walden and on the southeast by twelve miles of unbroken forest range, which includes the Groton State Forest.

There are several summer roads over the western and northern passes at about 1,800 to 2,000 feet. There has been only one road over the eastern range; at more than 2,000 feet, and impassable for cars in winter.

In 1896 there were doctors in each of the towns surrounding Cabot. Now there are none, and the nearest resident physician is at Plainfield, twelve miles away.

My boyhood home was in Walden, half a mile over the northern pass in the Lamoille watershed. I was the second of six sons of a poor farmer, on a good farm. When we were young, we attended eight weeks of school in the fall and twelve in winter. At the age of thirteen or fourteen we went to work out on farms to help support the family. We worked for our board and went to school during the twelve weeks in winter. I was given my time when seventeen years old. I then worked on a farm for my board and walked three miles to attend school about eighteen weeks in the fall and winter. The rest of the year I received wages. I taught school twelve weeks and worked on a farm two months from the age of nineteen until I was graduated from the medical college in this city (Burlington) in 1896.

I moved four miles down the valley to Cabot and have been an average country doctor for forty years. There were two other doctors in Cabot for a few years, then but one who remained in active practice until about ten years ago. Professionally, it is now a lonely life. It is not uncommon to go a month or two without seeing another doctor.

There were 638 doctors in Vermont forty years ago, one in nearly every village. Many of the country doctors were country boys who had paid or nearly paid their way through school and college. They commenced work when twenty-one to twenty-four years of age. If we take present conditions as a standard, they had a poor technical preparation, but they did know their people. The number in the state remained about the same until 1915. At that time there were 627 but in 1935 the number had been reduced to 415.

Towns now having hospitals had 243, and those with no hospitals had 395. In 1920 the number in each class of towns was 266. In 1935 hospital towns had 249 and the others 165. Hospital towns gained 29,000 population in thirty years and now have 147,000, or 600 to each doctor. The other towns lost 13,000 and now have 213,000 or 1,300 for each doctor.

Our Council on Medical Education was formed in 1904. The object was to secure fewer but better physicians for the work. We decided to kill 100 of the 160 medical schools. We were so brutal in our attack on men like myself and the colleges from which we graduated that we did not gain much. In 1908 we were joined by the great and powerful Carnegie Foundation. Another survey was made. Recommendations were made for the elimination of 120 colleges. Only two were to be left in New England. The added time and cost of preparation secured the desired result. The present graduate cannot afford to go to a small town. The small town doctors have moved away, died, or lost the confidence of the people. Their work is taken over

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†Burbank, L. W.—President, Vermont State Medical Society, 1935-1936. For record and address of author see "This Week's Issue," page 1313.

by the country merchant, the agent of a large corporation, the radio, magazines, and newspapers; also social workers from larger towns. The agent makes regular trips from house to house, finding out what ails the people and recommending a cure.

Eight hundred and forty-three Vermonters died of typhoid each five years from 1857 to 1897. From 1897 to 1901, 248 died. There were 125 deaths during the five years before 1921. Twenty-one died in the last five years.

From June to October we were busy caring for typhoid fever, cholera infantum, and digestive troubles of older people. The early reduction of deaths from typhoid was partly due to the use of better air, more food and cool sponging. The real advance was made by protecting the well folks from the excretions of the sick, and later, vaccination. The last death from typhoid in the town of Cabot was in 1905.

Diphtheria took 1,350 lives each five years from 1867 to 1897. Three hundred and sixty-five died during 1897 to 1901. There were 119 deaths from 1916 to 1921. The last five years the number was sixteen. We commenced the use of antitoxin in the early 1890's. Diphtheria was conquered, partly by the curative use of antitoxin; but mostly by isolation of the sick and the immunizing use of antitoxin and toxoid. The only diphtheritic deaths in Cabot have been from membranous croup. The last death was in 1912.

Scarlatina killed 587 each five years of the forty preceding 1897. From 1897 to 1901 the deaths were 89. The last five years they were 36. We have had three epidemics of the mild form in Cabot. Twice the severe form has started but was confined to a single family. There has been no death from this disease in Cabot for 40 years.

Forty years ago the sewage from the kitchen sink and the chambers was run into an open pool, just outside the kitchen window. There was an unscreened privy at the far end of the woodshed. The babies often took their unclean milk from an unclean nipple and through a long rubber tube, partly filled with curdled milk from a previous feeding. We did not know that flies carried the germs of disease, or that such germs thrived on the milk in that tube.

About 200 babies died each year of cholera infantum. Now the average number is 18. We soon had the people drain the pools, screen the privies and put a clean nipple directly on a clean bottle. During the war and soon after nearly all the homes were equipped with sanitary plumbing. Another disease was ended by protecting the well persons from the cause of that disease. The last death from cholera infantum in Cabot was in 1915.

So far as the saving of human life is concerned, the greatest advance has been made in the care of tuberculosis of the lungs. This disease killed 732 Vermonters each of the years 1857 to 1897. The average for 1897 to 1901 was 455; in 1920 it was 254. During the last five years the deaths have been 157 per year. When, in 1897, I first put a patient in an outdoor sleeping room, it was for the patient's good. I soon learned that the chief value of that plan was the protection of the rest of the family. Altitude, climate, diet, drugs and surgery have each had their turn before the spotlight. Each has helped some, but the greatest help has come from the isolation of the carrier.

There was a yearly average of 7,437 childbirths in Vermont from 1896 to 1900. The number was about the same until 1925. From 1925 the births rapidly decreased. The last five years there were only 6,348 per year. There were 8,102 in 1908 and 5,985 in 1935.

There were 40 puerperal deaths per year from 1897 to 1901; then 48, 51, 45, 37, 41, 39 and 35. During the first twenty years nearly all these confinements were in the homes. The poorly prepared family physician attended. During the last twenty years they have been in the care of well-qualified physicians and many of them in good hospitals. The average annual deaths for each thousand births is an index of the service given our women. They were, for the eight five-year periods, 5.3, 6.4, 5.6, 4.9, 5.4, 5.6, and 5.6. During the last five years more than 10,000 babies have been born in hospitals. Our best record was in 1934 when the death rate per 1,000 was 3.4. Last year, 2,300 of the 6,000 births were in hospitals and the rate was 6.5 per thousand.

The annual deaths from toxemia for the same periods were 12, 14, 15, 17, 17, 16, 14, 10.

The deaths from puerperal septicemia, a communicable and preventable disease, were 13, 12, 20, 14, 7, 8, 13, 11.

Deaths from hemorrhage were 5, 2, 4, 6, 8, 5, 6, and 5.

Deaths from all other causes were 10, 20, 13, 8, 6, 11, 7, 8.

The reports show about 25 deaths from cesarian section. Many of the doctors did not know in 1896 that they could carry puerperal septicemia from one woman to another. Neither did the domestic nurse know that she could carry it. The prospective mothers did not know the need of surgical cleanliness. We did not know that diet and medical care could prevent most of the severe toxemias. We did not have the pituitary preparations; the powerful oxytocic active principles of ergot had not been discovered. We could not, or at least did not, make use of blood transfusions. There were no cesarian sections. By special vote of the directors of Mary Fletcher Hospital, a woman in labor was admitted Nov. 29, 1902. Dr. P. E. McSweeney, assisted by Drs. Wheeler, Tinkham

and Arnold performed a cesarian section. The second birth in the hospital was another cesarian in 1905. The first normal case was in 1907. Last year there were 760 births in the hospitals in or near Burlington. Nearly every large town now has a good hospital where an increasing amount of maternity work is done.

Eight hundred and seventy children have been born in Cabot since 1896. Thirty-two have been born in hospitals, mostly in nearby cities. About twenty-five have been cared for in the home of Dr. and Mrs. Corson of Plainfield. I have attended 600 in Cabot—enough in surrounding towns to make a total of 871.

Ten years ago I reported to this society the deaths of all women in my care, who died between the sixth month of pregnancy and two months after confinement. Today I report only those classed as puerperal deaths by state and national health departments.

The first fatal case was after I had been in practice seven years. The woman was 23 years old, had been in the care of a specialist during pregnancy. She lived six miles over the mountains. The husband called me by telephone and said that his wife was "bloated big" and had been "blind as a bat" for two hours; and that her doctor was too busy to come. I insisted on counsel. She was in convulsions when we arrived. I gave her ether. The dead child was delivered by forceps. The room was kept dark and quiet. Eliminative treatment was commenced. When I went up the next day she was having convulsions about once each hour. The specialist had been there. Large doses of morphine were being used. I did nothing. The specialist was there that day and the next. I was sent for on the fourth day because her doctor could not attend her. She died a few minutes after I arrived. I signed the death certificate.

The next fatal case was one of my patients, a Para-VI, aged 29 years. She had no care during pregnancy. She had borne five children in rapid succession. Her pregnancies had been increasingly toxic. I had told her during her last pregnancy that she would not be likely to live through another. I did not know of the sixth pregnancy. When I arrived she had a blurred vision, was very edematous, and had an intense headache. The boiled urine looked like custard; the blood pressure was 220. When I said she had not been fair with me, she told me she knew that, but she did not want to live. She soon had a convulsion and did not again become conscious. The baby was unborn.

The other toxic death was that of a short, stout Para-IV, aged 27 years. She was tender in her right upper abdomen before and during her third pregnancy. She was nauseated, but did not vomit much. The skin was slightly bronzed during the last month. Labor was normal and recovery good. Two years later her hepatic symptoms returned. She consulted a surgeon who advised an operation. She did not follow the advice. Four years after the birth of her last child, she again became pregnant. Her toxic condition was worse than the first time. She could not afford a nurse. I would not care for her unless she did have one. A friend offered to help if she would go to a hospital. She went to the hospital, had twins and died. I report this as one of my deaths because I think she would have died at home.

I attended ten other women who had severe toxemias. Seven of them had convulsions. During the last ten years only two have been severely toxic. One recovered at home. The other had no place to be sick at home. She went to the hospital and had an ordinary labor.

During the first thirty years I cared for fifteen women who had severe hemorrhages. They all lived. There were four placenta praevias, one of which was marginal. A few times I had another doctor to help, but such cases cannot wait for help. Two of the placenta praevias were delivered by internal version. In one case, one man and one woman helped. The mother and baby both lived. One woman helped in the other case. My helper was six months' pregnant. There was not a clean cloth in the house. She brought in, from the clothesline, some clothes which had been boiled, washed and hung out to dry. The patient was in a dirty bed, in a pool of blood, and had fainted. There was a boiler full of boiling soapsuds on the stove. No ether was used. The mother and baby did finely.

I never saw the time when I would volunteer to care for a placenta praevia without the aid of another doctor and nurse. I have recognized two placenta praevias since 1926. One went to a hospital two weeks before labor. She did not recover. The other had several hemorrhages at home; then had cesarian section in a hospital. She made a slow but good recovery. She had a live baby.

There were two cases of puerperal septicemia. Both were in clean homes. Both had plenty of clean clothes. Both had normal labors. The domestic nurses who cared for them were clean and intelligent. One case was normal until the tenth day. Then she had a long severe sickness and recovered at home. She did not have any evidence of pus. The other case had a normal pulse and temperature when I saw her the third day, but was very tender over the left fallopian tube. She had a discharge of much pus the fifth day. She went to a hospital and recovered.

I generally see my patients the second or

third day. I tell them to come for an examination in a month. They do not come. When I am engaged for a confinement case, I make an examination. To prevent infection, the prospective mother boils, washes, dries in the sunshine, and irons sheets, pillow slips, towels, night clothes and a bundle of clean cloth. These are wrapped in clean paper and put in a bureau drawer. We try to have plenty of clean newspapers. I try to keep all these things free from dust or other things not clean.

The woman lies on her left side, with knees drawn up during the last efforts. She is covered with a clean piece of cloth about one yard square. Over that is a clean sheet. I wear a glove and an apron or gown. The hair is clipped. It is never shaved. I make a vaginal examination as often as is necessary, to keep informed as to the progress of labor. I never make a rectal examination. I would make them all rectal if I was where I thought there might be sources of infection. I try to have only one other person in the room. She is on the other side of the bed giving ether. If the perineum is torn to a slight or moderate degree, I put sterile gauze in the vagina, and place the stitches while the mother is on her side, and still partly under the influence of ether. The ends are left long and held by snaps. While I am placing the stitches, my helper is at my left. With a hand wrapped in sterile gauze she supports the flesh above my work. Stitches are tied after the placenta is removed.

During any other operative work, the patient is in the dorsal position. I try to make the caretaker know what I mean by keeping clean. If I do not dare trust her, a package of clean pads are placed where the mother can reach them. She changes her pads. I then go home feeling that if that woman has puerperal septicemia, it is because I carried it to her by means of my nose or throat.

There were 463 mothers and 860 confinements.

There were 871 babies; 843 of them were living and livable.

There were seven pairs of twins. Thirteen of these babies were living and livable.

One set of triplets was born at six months. They were born dead or died in a few hours.

There were 13 malformed babies. These were nearly all born dead or died in a few days or weeks. They were hydrocephalic or spina bifida. One had a perfect body and limbs, but no head. One had hydrocephalus and spina bifida. She is now living at three years of age.

Dr. Corson and I each had one case of ophthalmia neonatorum. Neither of us had any of the impetigo contagiosa group of diseases.

When necessary I have used high forceps, done internal versions and craniotomies. Twice I have done internal version after a hand has been born. One of the babies lived. On each occasion I had the assistance of two neighbors.

I have not told these personal experiences to show my crude ways, but for their bearing on the subjects of infection and immunity.

Dr. Corson of Plainfield has allowed me to use his records while studying immunity. He has been in Marshfield and Plainfield for about twenty years. He is clean in his obstetric work; and more modern than I in his methods. Dr. and Mrs. Corson have cared for about 225 births in their own homes. The doctor has cared for enough others in the patients' homes to make over 700. He has had only one mild case of puerperal infection. The only death in his series of home deliveries was one that was caused by hemorrhage. I went down to try and help him, but I could add nothing to his efforts.

We have probably had the usual number of abortions. Some are induced. During the early years, drugs were used. Oil of tansy, oil of cedar, and so forth, also fluid extract of ergot, were among those employed. The drugs frequently produced convulsions but the patients did not die. Then came an era of mechanical starters used by doctors in other towns. We then had many infections, but no deaths. For many years the woman or a friend has produced the abortion. Sometimes I am called to complete the operation. My call is generally on account of hemorrhage. If the patient is a multipara I use my hand or a dull curette. I always use a tampon for a primipara, unless I fear infection. There has been no death from abortion in the town of Cabot for forty years.

SUMMARY

So far as clinical puerperal septicemia is concerned, the child-bearing woman of the upper Winooski Valley, has for forty years been immune to all the germs in, on, or about her own body; and to most of those in her own home.

The doctors, who have practiced in the small towns of Vermont, have been useful members of the communities where they have lived.

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Case Records

ANTE MORTEM AND POST MORTEM

AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

AT THE

MASSACHUSETTS GENERAL HOSPITAL

Founded by RICHARD C. CABOT, M.D.
TRACY B. MALLORY, M.D., *Editor*

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22531

PRESENTATION OF CASE

A 26 year old male nurse was admitted complaining of cough.

Three days before entry the patient took a long automobile trip in an open car and that evening developed a nonproductive cough. There was no chest pain or chills. On the following day, however, he developed soreness along the costal margin on both sides, more on the right. Later this extended up into the right axilla and was described by the patient as a "catch in the side" while coughing. He felt chilly, but there was no rigor. On the day before admission he remained in bed and the cough was at this time accompanied by a small amount of blood-tinged sputum. The discomfort became localized in the right posterior axilla and was aggravated by coughing. He felt chilly and his temperature was 101.4°. On the day of entry his temperature rose to 103°, malaise became marked, and he had severe headache and anorexia. On the way to the hospital he became nauseated and vomited about a pint of greenish fluid.

Physical examination showed a well-developed, thin young man with flushed face lying flat in bed complaining of nausea and headache. The skin was warm and moist. The conjunctivae were moderately injected. The mucous membranes were dry. There was slight lessened motility of the right side of the chest and the right diaphragm appeared to be immobile. Resonance was diminished at the right base posteriorly and in the axilla. Breath sounds were likewise diminished, the respiratory phase being harsh in character. A few fine rales were audible in this region on deep inspiration. Spoken voice was not altered and tactile fremitus was normal. The remainder of the examination was negative.

The temperature was 102°, the pulse 126. The respirations were 32.

Examination of the urine was negative. The blood showed a red cell count of 5,200,000, with a hemoglobin of 80 per cent. The white cell count was 12,500; no differential count was recorded. Several specimens of sputum contained blood, and examination showed that the pneumococci present were not Type 1, 2 or 3. Sputum cultures showed alpha hemolytic streptococcus and *Staphylococcus aureus*. Repeated blood cultures were negative. A Hinton test was negative. The blood chlorides were 94 cubic centimeters.

A portable x-ray film of the chest showed dense homogeneous dulness involving the region of the left upper lobe. The remainder of the lung fields appeared clear.

The patient's temperature remained between 102° and 104° and the pulse was consistently elevated between 90 and 110. On the second hospital day dulness with increased tactile fremitus was elicited in the right upper chest anteriorly and in the axilla. There were exaggerated voice sounds and bronchial breathing in this region. The patient remained fairly comfortable although the physical signs remained unchanged. From the second to the sixth hospital day his white blood cell count remained in the vicinity of 5,000 and 2 days later coarse rales became audible over the right upper and midchest. At this time the white cell count was 15,000. His temperature became irregular and varied between 100° and 104°. There was slight cyanosis and rales became audible throughout the entire right chest, although no dulness or bronchial breathing was noted. The white cell count continued to rise and at the end of 2 weeks was 31,000 with 99 per cent polymorphonuclears. Rales at that time were audible on both sides, more prominently on the right and an x-ray of the chest showed considerable clearing of the consolidation in the right upper lobe. There were, however, fleck-like areas of consolidation throughout the entire right lung field and to a lesser degree in the left lung field. The patient's respirations became labored and cyanosis appeared. He was placed in an oxygen tent with slight relief, but his condition became progressively worse. On the sixteenth hospital day the white cell count was 44,000, with 94 per cent polymorphonuclears. Physical examination showed cyanosis and distressed breathing. Loud coarse rales were audible throughout both sides of the chest. Irregular areas of dulness and bronchial breathing were elicited bilaterally. There were no areas of suppressed respiratory sounds, and tactile fremitus was normal throughout. The blood pressure was 140/80. He became rapidly worse and died on the seventeenth hospital day.

NOTES ON THE HISTORY

DR. FREDERICK T. LORD: The temperature (looking at chart) is elevated but intermittent, not remittent, and tending downward. The pulse tends upward. It was pretty high all

through the illness. The respirations finally rose to sixty.

X-ray Interpretation

Dr. George W. Holmes: In the portable film, we have the characteristic picture of consolidation involving the region of the right upper lobe. That is quite typical and definite consolidation. Then 8 days later, the process has involved the entire right lung and I suspect the middle portion of the left, and this is characterized by a diffuse mottling. I am not certain about the cavity. In the record a cavity is mentioned and I think possibly that there is one present. This looks like the spread of a tuberculous pneumonia, a rapid spread of the disease after pneumonia.

Dr. Lord: I do appreciate your suggestion.

Dr. Holmes: That might be misleading, however.

Notes on the History Continued

Dr. Lord: The symptoms of onset are atypical for lobar pneumonia which ordinarily begins abruptly with cough, pain, bloody sputum, chill and rapid elevation of temperature, all within a few hours, and in this case they are nonexplosive. There are early, severe, constitutional symptoms here, but variation in this respect in individual cases of the same type of infection are common and thus no help is to be derived from this aspect.

The pulmonary process was at first lobar or massive. Resolution took place rapidly in this region, followed by the appearance of scattered, small, soft, mottled areas of increased density.

There was at first a moderate leukocytosis, then a low white count and later a marked increase in the white cells in the blood.

We would like to know more about the sputum, was it rusty or tenacious? With respect to the bacteriology, Types I, II and III pneumococci can be excluded, but a higher type might have been present. It is desirable to know about tubercle bacilli in the sputum.

Dr. J. H. Means: There were no tubercle bacilli present, Dr. Lord.

Dr. Lord: Though alpha hemolytic streptococci were found in the sputum, they are common in the respiratory tract, nonpathogenic and without special significance. *Staphylococcus aureus* is also a common finding in the sputum and I am not disposed to regard it as of special importance.

The x-ray findings are unusual, starting with a massive process, largely disappearing within 8 days, with the appearance of a patchy, diffuse involvement.

Now to proceed to what it might be. Is it infarction or infection? The upper lobe is an unusual site of infarction and, with an area of infarction of this size, we may expect frankly bloody sputum and more pleural pain. We may, I think, exclude infarction and then have left an infective process of some kind. Absence of preceding manifestations elsewhere puts it in the group of a primary pulmonary infection.

Differential Diagnosis

With respect to the cause, the different types of pneumonic infection do not present distinctive clinical pictures, and the attempt should be made to classify them etiologically and that, I suspect, is what I am supposed to attempt to do here.

In the absence of evidence regarding the etiologic agent, the best I can do is to enumerate the possible causes. Infection with a higher type of pneumococcus cannot be excluded. As already intimated, the alpha hemolytic streptococcus and the *Staphylococcus aureus* are of doubtful significance.

Regarding tuberculosis, the leukocytosis and the rapid resolution of the lobar involvement are against it. Initial massive tuberculous areas resolve, leaving scars and calcification, but only after the lapse of months. We may, I think, exclude tuberculosis. It might be plague pneumonia, but the duration is too long, the spleen should have been enlarged and, after all, we do not have plague here.

In addition, we have to consider two types of respiratory infection due to virus.

Is it influenza pneumonia? We must have interepidemic and interpandemic influenza to keep the virus alive. The leukocytosis is against uncomplicated influenza pneumonia and there was no preceding upper respiratory tract infection which occurs in 77 per cent of the cases. Experimental immunologic tests with the virus in animals would be necessary to prove it. I do not think influenza pneumonia can be excluded.

The second possibility is psittacosis and the inclusion with the x-ray films yesterday, when I saw them, of a paper with a notation regarding this disease leads me to discuss this possibility more at length. In psittacosis there is only a moderate or no leukocytosis, and the marked leukocytosis here is against uncomplicated psittacosis. There has been only limited opportunity for thorough study of pneumonic infections with this virus, but from such reports as are available, the lung involvement is usually massive, as in the upper lobe process here, and if spread takes place, it occurs with the development of fairly large focal processes unlike the numerous small scattered areas in this case. Patchy consolidation, however, was observed in the proved case reported by Sprunt and Berry. It would be desirable to know of exposure to

birds and especially parrakeets, or to other cases of the disease. To establish this as the cause, it would be desirable to investigate the blood or the sputum by passing the latter through a Berkefeld filter, the inoculation of mice intraperitoneally and the finding of psittacosis bodies in smears from the peritoneal exudate, and establish the presence of an immunity against a known strain of the virus in the recovered mice. It is of some interest in this connection that investigations during the life of the patient are more likely to be successful than on the cadaver.

Finally, it seems to me that the only justifiable conclusion, with the evidence at hand, is that he had an atypical pneumonia of unknown origin and that it may fall into one of the groups I have discussed.

DR. TRACY B. MALLORY: Dr. Means, you might explain how the question of psittacosis came up in this case.

DR. MEANS: I saw this patient only once. It was the day before he died. Dr. Jones was away and I was asked to take the visit. The patient was in an oxygen tent, was moribund, had deep cyanosis, and presented the picture of a very septic type of diffuse bronchopneumonia. We looked at the x-rays and thought it amazing that he should first have a picture which seemed to be lobar pneumonia, anatomically clearing up, to be followed by one of bronchopneumonia of which he died. We did not have sufficient imagination to think of psittacosis at that time, nor at the autopsy, nor did the pathologist suggest it. The matter of psittacosis arose some time later when another nurse, a man of about the same age who was a friend of this one and who had been with this one, came in with a disease which bore some points of resemblance to this case and some points of difference. This second man, who is now well, presented the picture of slowly evolving pneumonia which involved the right lower lobe incompletely and was characterized by leukopenia from the beginning, with very little intoxication, slight splenomegaly, and a course that clinically resembled mild typhoid rather than pneumonia. He went through a febrile period of about 10 days and defervesced by lysis uneventfully. He never had a frank lobar consolidation but had a lot of rales at the right base which finally disappeared. When this latter individual was at the height of his fever we got worried and wondered if both these patients had some strange disease which in the first one ran a fatal course. We did not know what it was. We talked with Dr. Dienes and I think he was the first man to suggest that both cases might be psittacosis. It turned out that neither man had been near a parrot since August when they both had been to a zoo and had walked by parrots but had not fondled them. That was all we could do in the parrot line. Dr. Mallory went over the sections on the first case carefully at that time to see whether anatomically they bore any resemblance to the type of lesion described in psittacosis. We found that they did not. We do not know, therefore, whether the first man had psittacosis or not. We were inclined to believe that he did not, and I am sure now that the second did not.

Dr. Lord asked a question about the sputum in this case. I saw the sputum and it was not in the least like that of lobar pneumonia, not rusty or tenacious; it was watery, full of pledgets of pus and a few blood streaks. I saw a great deal of influenza during the War and it did not resemble either of these cases, but they may be influenza nonetheless, presenting a different picture because the secondary infection was different from what we were dealing with in 1918 and 1919. Dr. Russell made ward rounds with us and saw the second man. He was interested and thought that the leukopenia suggested a virus disease, very likely influenza, and suggested that we get some serum from all the cases of this type and also some convalescent serum from this man and send them to the International Health Division for Dr. Frances to determine any anti-influenza activity which might be present. That is being done.

DR. WYMAN RICHARDSON: I might add that aside from these two men there was one other nurse in the same hospital who took care of one of them and he was very sick for three or four days but recovered uneventfully. He had no great increase in white count and a few signs at the left base with rales. Then there were two more men among the same group of male nurses who ran a fever for 4 or 5 days and recovered without complication.

CLINICAL DIAGNOSES

Septic bronchopneumonia.
? Influenzal bronchopneumonia.
? Pulmonary tuberculosis.

DR. FREDERICK T. LORD'S DIAGNOSIS

Atypical pneumonia of unknown etiology.

ANATOMIC DIAGNOSES

Bronchopneumonia with abscess formation, staphylococcus.
Meckel's diverticulum.
Acute cystitis, slight.
Operation wound, appendectomy.

PATHOLOGIC DISCUSSION

DR. MALLORY: Of course three cases of pneu-

monia do not necessarily constitute an epidemic but when they occur in close proximity, physically and temporally, one immediately becomes suspicious of the possibility, and the number of causes of epidemic pneumonia are relatively few. It was for that reason that both psittacosis and influenza were very seriously considered in this case. I am not sure that the autopsy findings give us any answer. We found at the time of postmortem examination that this right lung which had been originally involved had practically cleared up, whereas the left lung at the time of autopsy showed much more extensive involvement through the lung. This consisted of multiple abscesses and direct smears from these abscesses showed innumerable cocci with the characteristic arrangement of staphylococci. On culture it turned out to be a *Staphylococcus albus*.

The other findings at autopsy were a septic spleen which contained rather a larger number of mononuclear phagocytes than usual and a tremendous degree of hyperplasia of the bone marrow, so that at first glance one would actually think of leukemia. There are even a few very small foci of myelopoiesis in the liver. There was no true leukemic infiltration, however, and no myelopoiesis was found in the spleen. I believe the depth of the infection is an adequate explanation of the bone marrow findings.

I think we have to assume in this case an unknown primary contagious infection followed by a secondary pneumonia. We have no evidence as to what the etiology of the primary infection may have been. In the second case we did inoculate mice in order to rule out psittacosis. At what stage this staphylococcus came into the picture, I do not believe we can be sure. We can be confident it was not the primary infection. It may have been the secondary or may even have been the tertiary infection. During life he had three blood cultures of which two were negative and one showed a *Staphylococcus aureus* in one of two flasks but since the second flask inoculated at the same time was negative, that was interpreted as a contamination and therefore was not included in the record. It may have been significant.

DR. LORD: I would like to ask Dr. Holmes if he would agree about my interpretation of the spread of this being against tuberculosis?

DR. HOLMES: Yes, but I think you can interpret that another way. If a patient had quiescent tuberculosis at the apex and an acute lobar pneumonia, as the pneumonia resolved there might be a breakdown and spread of the tuberculosis; so that I think it would be perfectly possible to interpret this picture as being due to tuberculosis.

CASE 22532

PRESENTATION OF CASE

First Admission. A 20 year old American girl was admitted complaining of a draining sinus in the left chest.

Eleven years prior to entry the patient had an attack of bronchopneumonia following which she continued to have cough productive of light-colored mucoid material amounting to about a half a cupful a day. There was some dyspnea on exertion but no orthopnea. About 2 years before entry the patient lost 10 pounds over a period of several months, became tired, weak and felt listless. X-ray examination showed fluid in the left chest and she entered a hospital where a tube was inserted into the pleural cavity. She remained in the hospital for 18 weeks and was kept in bed during that time for 3 months. She gained strength and was finally discharged with the tube still in place. It was not until 5 months after discharge that the tube was removed and following this there persisted a sinus tract which drained light yellowish fluid which occasionally was purulent in appearance. Pads were worn over this orifice and these required changing two to four times daily. Except for the inconvenience of the sinus the patient felt quite well and led an active existence.

The family history is noncontributory.

Physical examination showed a thin underdeveloped girl lying flat in bed in no discomfort. She had an occasional nonproductive cough. There were a few shotty cervical nodes on the right side and some depression of the supraclavicular fossae. The trachea seemed to be in the midline. The entire left chest was dull anteriorly and posteriorly and the breath sounds were diminished to absent. Occasional crepitant rales were audible at the left base. Tactile fremitus and vocal resonance were diminished. On the right side the physical signs were normal. The cardiac impulse was felt in the right midclavicular line and the heart sounds, which were of good quality, were best heard in this region. No murmurs were heard. The tip of the spleen was palpable just beneath the left costal margin.

The temperature was 99.5°, the pulse 120. The respirations were 25.

Examination of the urine was negative. The blood showed a white cell count of 21,400, with a hemoglobin of 75 per cent. A smear of the material from the chest sinus showed many gram-positive diplococci with capsules. Cultures gave no growth.

X-ray examination of the right chest was negative. The entire left chest exhibited homogeneous dulness, more dense at the base than at the apex. The heart shadow was slightly displaced to the right but there was no displacement of the trachea. The interspaces on the

left were slightly narrowed and there was slight scoliosis. In the first interspace was a 2 centimeter round area of decreased density which had the appearance of a cavity with a fluid level. The trachea and the bronchi were well visualized and showed no gross variations from the normal. In the left lateral view there was no evidence of collapse of the lower lobe. The gas bubble of the stomach was not displaced. On deep inspiration there was a very slight shift of the mediastinum toward the dull side.

A thoracic paracentesis produced 14 ounces of foul purulent material. After one month, x-ray examination showed a fluid level just above the diaphragm. The pleura was separated from the axillary border and there was gross dulness at the apex. The findings were considered to be significant of a large cavity with fluid occupying the entire right lung field. The pleural cavity was irrigated through the sinus tract for 3 days, during which time her temperature fluctuated up to 103°. Subsequently the eighth rib was partially resected and the empyema drained. Directly afterward the temperature dropped to normal and remained at that level throughout her hospital stay. Eleven days after operation another x-ray film showed reaccumulation of the fluid in the lower left cavity. Separation of the parietal and visceral pleura along the left axillary border was increased only slightly since the last observation. The fluid level noted in the upper chest was still present. The patient improved rapidly and was discharged on the thirteenth hospital day.

Final Admission, 1 year later.

The patient had been followed in the Outpatient Department, where she continued to improve and her weight increased. The drainage tube was kept in place and drainage of purulent material continued. X-ray examination 8 months after discharge showed that the dulness in the left side of the chest was considerably reduced. There was no evidence of undrained fluid. The pleura was markedly thickened and the lung irregularly expanded. There was an area of dulness in the region of the right middle lobe which was thought to be possibly due to a collapsed bronchiectatic lobe. The patient returned to work as a clerk and felt perfectly able to perform ordinary activity without fatigue. Three weeks before reentry she had a cold of moderate severity which subsided without complication. There was no chest pain, hemoptysis, dyspnea or night sweats. Her appetite was good and her bowel movements regular. The wound still drained a little and she entered the hospital for an obliterative operation.

Physical examination showed a fairly well-developed and nourished young woman in no discomfort. There was a draining sinus in the left posterior axillary line at the level of the eighth rib. There was dulness posteriorly and laterally over the apex on the left side and impaired resonance over the remainder of that side. Breath sounds and voice sounds were diminished rather generally except at a point just medial to the tract where some resonance was elicited and breath sounds were bronchovesicular in character. No rales were heard. The heart was negative. The blood pressure was 124/80. The remainder of the examination was negative.

The temperature, pulse and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 5,300,000, with a hemoglobin of 90 per cent. The white cell count was 13,600.

X-ray examination of the chest following the injection of lipiodol through the sinus tract showed an elongated cavity, the lower portion of which was filled with the lipiodol. The cavity, however, appeared to extend upward above this along the lateral chest wall to the level of the clavicle. The opaque medium did not enter the bronchi.

On the fourth hospital day the fourth, fifth, sixth and seventh ribs on the left side were partially resected and the pleura underlying them excised. A section of the latissimus dorsi was implanted within the pleural cavity. A smear from the empyema cavity showed gram-positive cocci in pairs and gram-negative bacilli. A culture showed fluorescent gram-negative bacilli. Following operation the temperature rose to 103°, the pulse to 110 and the respirations to 23. Throughout the remainder of her hospital stay her temperature continued to fluctuate irregularly up to 103°. On the ninth postoperative day an x-ray examination showed that the entire left lung field was dull except for a small area at the apex. No fluid level was visualized. The heart was not displaced. The patient responded well but complained of soreness in the chest wall. On the twentieth day the tube was removed and there was very little discharge. A month after entry the patient developed cough with which she raised about 1 to 2 ounces of colorless sputum daily. The wound appeared to be infected and was treated by dakinization with improvement. Two months after entry, however, a revision of the wound was performed. The intercostal muscle bundles were found to be thick, pale and edematous. These were resected and a Dakin's tube placed in the wound. Her condition improved somewhat and her temperature subsided but 2 weeks later irregular fever again occurred. The patient became irritable and restless and complained of continuous headache. Another x-ray of the chest at that time showed dulness along the lateral chest wall on the left side as previously described. There was now air between this and the slightly retracted lung but no evidence of retained fluid.

The heart was slightly displaced toward the right. The rib stumps showed evidence of marked osteoporosis but no definite evidence of osteomyelitis could be discerned. A neurologic examination showed early bilateral papilledema. The visual fields were normal. There was no nystagmus, visual weakness or other localizing cranial signs. The reflexes were hyperactive but symmetrical. A lumbar puncture showed clear colorless fluid with an initial pressure of 205. The dynamics were normal and the final pressure after removal of 10 cubic centimeters of fluid was 95. The total protein was 154 milligrams, chlorides 618, spinal fluid sugar 43, and a Wassermann test was negative. The cell count showed 61 white blood cells, 50 per cent polymorphonuclears and 50 per cent lymphocytes and monocytes. A few days later another neurologic examination showed clonus of the left ankle and a Babinski sign on the left side. Further spinal punctures revealed gradual increase of cells to 370 leukocytes. The total protein rose to 190 milligrams. A third nerve paralysis developed on the left side with dilatation of the pupil, lidlag and inability to contract the superior, inferior or internal rectus muscles. The patient became progressively weaker, drowsy and her speech appeared impaired. She gradually lapsed into coma and died 4 months after reentry, 1 year and 4 months after the initial entry.

Differential Diagnosis

Dr. Richard H. Sweet: This is a girl of twenty who 11 years before entry, at the age of 9, had an attack of bronchopneumonia following which she had a cough productive of light mucoid material amounting to half a cupful a day. She had bronchopneumonia followed by a chronic pulmonary lesion which, of course, would make us think of bronchiectasis, possibly tuberculosis, or some chronic pulmonary disease. But about 2 years before entry she had another attack, characterized by loss of weight, when she felt tired, weak and listless. There is no story of pain, exacerbation of cough, or change in the character of the sputum.

She had pleurisy with effusion. We do occasionally see empyema of a very chronic sort following pneumonia which may go a year or more without a diagnosis being made; but I doubt very much if this episode had anything to do with her previous bronchopneumonia except that it might be secondary to the residual process following bronchopneumonia. Of course patients developing spontaneous pleurisy with effusion make one think of tuberculosis. The fact that she was operated on does not rule it out because I have seen patients operated on erroneously who had tuberculous effusion. They kept her in bed for a surprisingly long time, 3 months. In an ordinary pyogenic empyema the patient should be up after 2 or 3 weeks or less. She was in the hospital for about a month longer after getting up.

There is no note of what the x-ray showed at that time. It would have been a help to us. It strikes me as peculiar that she was in bed so long and also after having her tube in place for 9 months that she continued to have a persistent sinus. Of course, if this is an ordinary pyogenic empyema, the sequence of events is not uncommon. But one of the commonest causes of chronicity in a case of empyema is tuberculosis. In other words, many times we see patients who come in with a postpneumonic empyema, definitely proved to be such, which has become chronic and we do a thoracoplasty to give better drainage, and by doing a biopsy on the pleura we commonly find a tuberculous process. So I am always suspicious of any long-standing chronic empyema being tuberculous although other possibilities are foreign bodies, failure to leave the drain in a sufficient length of time, or inadequately placed drainage.

The chest signs are consistent with fluid or with a thickened pleura in addition. She may well have had a large spleen secondary to the chronic sepsis.

Culture showed no growth. This may not mean anything. It may not have been planted immediately.

The x-ray examination at this time showed that the right chest was negative. The entire left chest exhibited homogeneous dulness, more dense at the base than at the apex. All of these facts suggest possibly some fibrosis or atelectasis of the upper portion of the lung. This is the first note of any x-ray evidence of disease in the lung itself, so that we see definitely by the x-ray report that the lung and pleura were involved.

Dr. George W. Holmes: This film taken before operation shows a rather characteristic appearance of fluid. It is interesting that the lung on the other side is perfectly normal. There is no evidence of fibrosis. After operation the edge of the partially expanded lung shows some pleural thickening with possible fluid at the base. The heart is in the normal position. A film taken after injection of an opaque mixture into the sinus failed to show evidence of communication with the bronchus.

Dr. Sweet: The cavity is described as being in the lung field. Does that show here? In the first interspace there is a two centimeter round area of increased density according to this record. That area there has no lung markings. Is it fair to assume that that is a pulmonary cavity, or is that fluid?

Dr. Holmes: I suppose you are thinking of tuberculosis. I do not believe that cavity is due to tuberculosis.

DR. SWEET: They obtained 14 ounces of foul purulent material. After a month the x-ray showed a fluid level above the diaphragm. The pleura was separated from the axillary border.

"The patient improved rapidly and was discharged on the thirteenth day." That puzzles me because as we read along, in spite of the fact that she had reaccumulation of fluid, we find she went home feeling much better. I presume the tube became plugged up and the empyema backed up. She was discharged presumably in good condition although we have no note as to how she was or of the amount of sputum, or even of the presence of sputum. There is a history of dulness in the region of the right middle lobe which could be due to collapsed middle lobe.

DR. HOLMES: In going through the films I did not correlate them.

DR. SWEET: So now we have a picture of chronic empyema given adequate drainage at the second operation here at this hospital, the patient having done well with the exception that the empyema had not healed. My first impression that she had an intrapulmonary process in addition to a pleural process seems to be erroneous as demonstrated by the x-ray film. The physical signs were consistent with an old empyema cavity. As Dr. Holmes pointed out, there was no bronchial communication and the lung showed no involvement.

DR. HOLMES: I suppose I should have said no bronchial communication was demonstrated. There is no evidence of it.

DR. SWEET: "On the fourth hospital day the fourth to seventh ribs on the left side were partially resected and the pleura underlying them excised, and so forth." That is an operation which we do sometimes for persistent chronic bronchial fistula after lung abscess and sometimes for chronic empyema. The x-ray I thought was going to show an empyema cavity going up as far as the clavicle as they described it, but apparently it was localized and that explains the apparent discrepancy between the report of the x-ray and the size of the operation.

Obviously she had a septic wound following the operation. That is the first thing you would think of under any circumstances. This matter of sputum has been very difficult for me to follow throughout this whole history. At one point in the first paragraph we might assume she had sputum throughout the whole history but during the period of observation in the Outpatient Department no mention is made of sputum. Now we have mention of sputum 1 month after the operation which would suggest that something new had happened, something involving the lung.

Of course that first rapid rise in temperature, pulse and so on, showing evidence of infection, may have been due to the intrapulmonary process which she must have had, because she developed a sputum; or it may have been due to the wound. I would personally be very suspicious of the wound. The intercostal bundles at this revision of the wound were thick, pale, and edematous. I do not believe that means anything. The temperature subsided after operation so the trouble was apparently in the operative wound. If she had something in the lung, abscess, bronchopneumonia, or something of that sort without external drainage I should think it would have continued and the temperature and signs of infection would not have subsided; but now we have another aspect to the case.

"Her condition improved somewhat and her temperature subsided, but 2 weeks later irregular fever again occurred." Apparently the house officer or someone had thought the patient had osteomyelitis of the ribs to explain this fever.

With the headache and with this early bilateral papilledema we must begin to suspect a complicating cerebral lesion. Of course a metastatic brain infection from pulmonary or pleural disease is relatively frequent. I am not particularly expert at interpreting these figures on the lumbar puncture, but I should think they would be consistent with a reaction in the spinal fluid to a neighboring infection. Or they might go with tuberculous meningitis although the white count is not typical.

I presume there is no particular need to make this case very complicated. The only thing is to explain why she developed this empyema 9 years after having had bronchopneumonia. We have no knowledge as to what she did during the interval between the bronchopneumonia and the development of empyema except that she had a cough with sputum. The empyema came on rather gradually. At any rate our first definite knowledge of her shows her as a case of chronic empyema without pulmonary damage, which is different from my first impression. But assuming that that is the case, then the rest of the picture becomes rather clear. She had this chronic empyema and following the operative attempt to obliterate the cavity things went badly. She had infection in the wound, and may have had infection in the lung following the operative interference, with finally a metastatic involvement of the brain and, I should presume, brain abscess.

DR. TRACY B. MALLORY: I think we ought to give Dr. Sweet one additional piece of information, one which his discussion shows he would have looked for and one which was given to the surgeons but they refused to take it. On the occasion of this revision of the operative wound

they excised the intercostal muscles and we found in them foci of calcification and a great many collections of foreign body giant cells in arrangement so suggestive of tuberculosis that we reported it flatly as tuberculosis. The surgeons then came over and said that it could not be tuberculosis and asked us to reconsider our diagnosis. We reconsidered, lost our courage a little bit, and said perhaps it was not tuberculosis, but that we would like to have a little more material to put into a pig.

DR. SWEET: Before I started the discussion my diagnosis was tuberculous empyema with tuberculous meningitis. There are many things consistent with that diagnosis, some of which are the failure of operation, the persistent empyema which is so common with tuberculosis, and the failure of thoracoplasty which is almost invariably successful except in the presence of tuberculosis. I was swung from that diagnosis by the x-ray which seemed to prove the absence of evidence of tuberculosis, but with even suggestive histologic support I prefer to return to it.

CLINICAL DIAGNOSES

Chronic empyema.
Brain abscess.
Infection of chest wall.

DR. RICHARD H. SWEET'S DIAGNOSES

Tuberculous empyema.
Metastatic tuberculous meningitis.

ANATOMIC DIAGNOSES

Tuberculous empyema.
Tuberculosis, pulmonary, healed apical.
Tuberculosis of bronchial and retroperitoneal nodes.
Meningitis, tuberculous.
Pulmonary atelectasis.
Congenital absence of the middle lobe of the right lung.
Parovarian cysts, left.

PATHOLOGIC DISCUSSION

DR. MALLORY: We found at autopsy no evidence of a cavity in the lung. There was, however, old healed tuberculosis at the left apex and there were in the lower portions of the left lung some fairly fresh tubercles. There were large caseous hilar glands. We also found scattered miliary tuberculosis in the liver and spleen and basilar tuberculous meningitis without any solitary tubercle in the brain; so that the involvement of the basal cranial nerves was presumably due to the meningeal involvement.

DR. HOLMES: Was there any evidence of tuberculosis in the contralateral lung?

DR. MALLORY: No.

DR. SWEET: One of the things that I have been tremendously impressed by is the frequency of tuberculosis in chronic empyema in the absence of demonstrable tuberculosis. This case is exactly in line with a number of cases I have seen.

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PRONTYLIN AND PRONTOSIL

THE newspapers are crediting "prontylin" and "prontosil" with power to overcome the effects of the invasion of the human body by streptococci.

These drugs are "produced by a linkage of azo dyes with a sulphonamide group". Prontylin is intended for peroral use and prontosil is administered by the intramuscular route or directly into the peritoneal cavity according to the special indications. Prontosil is the more powerful of the two.

According to the published reports these drugs are efficacious in certain streptococcic infections and to a lesser degree in combating staphylococci. Experiments have also been made in the use of these drugs in other specific infections. It has been stated that they are not effective against *Streptococcus viridans*. Credit for the demonstration of the efficacy of these drugs has been given to Domagk, a disciple of Professor Heinrich Hoerlein, director of pharmaceutical research for the Interessengemeinschaft.

The results of Domagk's studies were published in 1935[1] and the early application of his work was carried on by German and English research workers and clinicians. In the United States, studies have been made of the use of these drugs in many places, especially at Johns Hopkins, the Presbyterian Hospital in New York and several hospitals in Boston.

Published clinical results are far from convincing to one that is inclined to be skeptical. According to Colebrook and Kenny[2] the articles in the German literature do not state whether all cases were infected with hemolytic streptococci, whether blood stream invasion was present or whether serious complications, such as peritonitis, existed. These English authors[2] report on the treatment with prontosil of thirty-eight cases of puerperal sepsis. Only three cases died, a mortality of 8 per cent, whereas the expected mortality, based on statistics of the immediately preceding years, was 22 per cent.

It must be borne in mind that the virulence of streptococci varies tremendously from year to year, even from month to month, and that the only way to evaluate properly a therapeutic agent of this sort is by the "alternate case method". Furthermore, only six of the cases in this series had hemolytic streptococci in the blood stream and, of these, three died, a mortality of 50 per cent. It should also be noted that some of the patients showed transient toxic effects to the drug and that three developed sulphemoglobinemia.

There is no report of consideration of the efficacy of these compounds by the Council on Pharmacy and Chemistry of the American Medical Association at the present time, and it would be well for the medical profession to leave further trial of them to competent observers even though statements of reputable physicians are impressive.

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REPORT OF THE SPECIAL HEALTH COMMISSION

As a result of the report of the State Health Commission appointed in 1935 by an act of legislature, far-reaching revisions of this State's health laws will be sought at the coming session

of the Legislature. The first section of the special commission's report has recently been made public by the Commissioner of Public Health, and more will be forthcoming.

Recommendations will be made that uniform state-wide regulations for the isolation and quarantine of communicable diseases be adopted, that unreasonable restrictions on burials following deaths from communicable diseases be abolished, that health departments be given authority to control persons suspected of spreading infection, and be held responsible for the conduct of adequate programs for vaccination against smallpox and immunization against diphtheria. It will also be recommended that the control of venereal diseases be made a state, rather than a local, function.

The control of tuberculosis takes up a major part of this first section of the commission's report, with many recommendations concerning it. One of the most important of these has to do with the regular examination of school teachers. It is furthermore recommended that county tuberculosis sanatoria eventually be placed under state supervision, with an ultimate discontinuance of all municipal tuberculosis hospitals except the hospital in Boston.

It may be pertinent, in discussing this report, to direct some attention to the manner in which it came into existence; to point out that it is the result of a survey consuming over a year's time and covering every important point with a bearing on the public health, made by an unpaid commission of several scores of interested and loyal professional individuals, largely physicians.

The labor was monumental, exact and inclusive, yet the task was so well organized and coordinated, with committees and subcommittees, that no undue burden was thrown on any group or on any individual.

The impressive fact concerning it is that in a period of more or less gross materialism, when few political acts are performed without some ulterior motive, a group of citizens should so willingly give of their time and of their ability to a valuable performance carrying with it no reward and no opportunities for self-glorification. The Massachusetts Medical Society should be congratulated that its membership figured so largely in this public-spirited enterprise.

CRIMINAL ABORTIONS AND REPUTABLE PHYSICIANS

THE activity of the criminal abortionist is a social menace and against it the statutes of the Commonwealth offer a certain amount of protection by prescribing punishment for the offender, if convicted. How widespread the activity is, no one can tell accurately, but the effects in morbidity and mortality are worth careful study.

It is a disturbing social phenomenon that so few criminal abortionists are convicted in court, yet the reasons are not hard to find. The pregnancy is an unwanted pregnancy and relief from the pregnancy brings relief from social problems which may prove in the highest degree embarrassing. The woman is not likely to repay her deep obligation, as she regards it, by doing anything that will cause trouble for the person who has helped her, and often if trouble comes to herself she will say, on advice, that the abortion was self-induced.

When the unmarried daughter in a family is about to die or has died, as the result of the offices of a criminal abortionist, there is often furious indignation that the murderer should be permitted to escape. Yet even such slight evidence against the criminal as the family may have is often withheld, because, they say, they do not want the publicity and it will not help the daughter who is gone. Even the argument of possible protection to other daughters is of no avail.

It is noteworthy that many of these women come under the care of a reputable physician before death finally closes the incident. What is the duty of the physician?

First of all, of course, it is to do his utmost to restore the patient to health, if possible. But is that all? Has he no duty as regards the person who committed the crime? Does his consecration to the care of the sick include protection to the criminal? Would it not be at least part of preventive medicine to assist in protecting other persons?

The harm done by the criminal abortionist has reached such proportions that there are some persons who advocate the compulsory reporting by physicians of all abortions, just as every gunshot wound is required by law to be reported, if the patient comes to a physician for treatment.

It may be that the compulsory reporting of abortions would not be necessary or wise or even expedient. But at the present time there seems to be a widespread indifference on the part of reputable physicians, in the matter of preventing criminal abortions, for they offer no assistance looking toward the apprehension of the abortionist. Would it be too much to ask that, in case a criminal abortion is suspected, the physician consult the medical examiner promptly instead of waiting until the death of the patient? It may be that no lives could be saved by calling the attention of the medical examiner to these patients when once blood-poisoning has gained control of the body, but if even a few other deaths could be prevented by checking the abortionist, a distinct service to humanity would be rendered.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

WHITE, PAUL D. A.B., M.D. Harvard University Medical School 1911. Physician, Massachusetts General Hospital. Lecturer in Medicine, Harvard University Medical School. His subject is "The Study and Treatment of Heart Disease at the Massachusetts General Hospital from 1821 to 1936." Page 1261. Address: Massachusetts General Hospital, Boston, Mass.

MASSELL, BENEDICT F. A.B., M.D. Harvard University Medical School 1931. Resident Physician, House of the Good Samaritan. Assistant in Medicine, Harvard University Medical School, and Beth Israel Hospital. Graduate Assistant in Medicine, Massachusetts General Hospital. Address: 25 Binney Street, Boston, Mass. Associated with him is

JONES, T. DUCKETT. B.A., M.D. University of Virginia School of Medicine 1923. Research Director, House of the Good Samaritan. Assistant in Medicine, Massachusetts General Hospital. Instructor in Medicine, Harvard University Medical School. Address: 25 Binney Street, Boston, Mass. Their subject is "Evaluation of the Signs of Active Rheumatic Fever: with Especial Reference to the Erythrocyte Sedimentation Rate and Leukocyte Count." Page 1269.

TAYLOR, GRANTLEY W. A.B., M.D. Harvard University Medical School 1922. F.A.C.S. Assistant in Surgery, Harvard University Medical School. Assistant Surgeon, Massachusetts General Hospital. Associate Staff Surgeon, Palmer Memorial Hospital. Surgeon, Collis P. Huntington Memorial Hospital. Visiting Surgeon, House of the Good Samaritan and Pondville Hospital. Consulting Surgeon, Massachusetts Eye and Ear Infirmary. His subject is "Carcinoma of the Breast in Young Women." Page 1276. Address: 264 Beacon Street, Boston, Mass.

INGALLS, THEODORE HUNT. A.B., M.D. Harvard University Medical School 1933. Medical House Officer, Peter Bent Brigham Hospital, 1934-1935. William Hunter Workman Fellow of Harvard University Medical School. Research Fellow in Pediatrics, Harvard University Medical School and The Infants' and The Children's Hospitals, 1935-1937. His subject is "The Role of Scurvy in the Etiology of Chronic Subdural Hematoma." Page 1279. Address: 300 Longwood Avenue, Boston, Mass.

CASEY, JOHN F. M.D. College of Physicians and Surgeons, Columbia University 1909. Visiting Physician, St. Elizabeth's Hospital. Consulting Physician, Leonard Morse Hospital. His subject is "Brucellosis (Undulant Fever)—Interesting and Important Facts About the Disease, with the Report of a Severe Case Occurring in a Boston Physician." Page 1282. Address: 475 Commonwealth Avenue, Boston, Mass.

KUGELMASS, I. NEWTON. M.A., Ph.D., Sc.D., M.D. Yale University Medical School 1925. Director, Pediatric Research, Fifth Avenue Hospital, New York. Director, Heckscher Institute for Child Health, New York. Associate Attending Pediatrician, Fifth Avenue Hospital, New York. Attending Pediatrician, Broad Street Hospital; French Hospital; New York City Children's Hospital. Consulting Pediatrician, Monmouth Memorial Hospital, Long Branch; Lynn Memorial Hospital, Sussex; Muhlenberg Hospital, Plainfield, New Jersey. His subject is "Milk Modification and Infant Constitution." Page 1285. Address: 1060 Park Avenue, New York City.

BURBANK, L. W. B.S., M.S., M.D. University of Vermont College of Medicine 1896. President, Vermont State Medical Society, 1935-1936. His subject is "Medical Service in Vermont: Some Changes in Forty Years." Page 1292. Address: Cabot, Vermont.

MISCELLANY

CONNECTICUT NEWS

STANDING ORDERS AND POLICIES FOR PUBLIC HEALTH NURSES

This pamphlet appears as a revision of "Guidance for Public Health Nurses" and was endorsed and approved by the House of Delegates of the Connecticut State Medical Society September 23, 1936. These standing orders should insure adequate nursing care in any community since they provide for nursing care in families where no physician is in attendance or where the attending physician has left no orders for the nurse. They also provide a guide in health supervision services as afforded by the public health nurse during periods of maternity and infancy and in public health education for preschool and school children and adults. As far as is known Connecticut was the first state where the Department of Health and the Medical Society gave joint approval to public health nursing orders and policies.

WATER AND SEWAGE IMPROVEMENTS

On October 7, 1936 a new chlorination plant was placed in service on the Jewett City Company system. This supply was one of the largest untreated surface waters in the state, furnishing water to approximately 3,000 persons. The Unionville Water Company installed temporary chlorination on its untreated surface supply on September 4, 1936, in anticipation of new permanent chlorinating equipment now being installed. Several other chlorinator in-

stallations are under way. The new sewage treatment plant at Uncas-on-Thames, State Tuberculosis Sanatorium, was placed in operation on October 5, 1936. This plant comprises coarse bar screen, sedimentation tanks of the Imhoff type, glass-covered sludge drying beds and chlorinator.

Connecticut Public Health Association

The Connecticut Public Health Association met in Waterbury on December 16, 1936. The morning session was given up to a symposium on pneumonia and included among the speakers Francis G. Blake, M.D., Professor of Medicine, Yale University School of Medicine, and Joseph I. Linde, M.D., Health Officer of New Haven. In the afternoon syphilis was considered with R. A. Vonderlehr, M.D., Assistant Surgeon General, U.S.P.H.S.; John L. Rice, M.D., Health Commissioner, New York City, and several local authorities as speakers. The talkie movie "For All Our Sakes," previously reviewed in Hartford, was presented at the close of the program.

Outbreak of Bacillary Dysentery

Bacillary dysentery recently appeared in an institution in Hartford. Forty-two inmates were afflicted and the duration of illness varied from 1 to 4 days. Paradysentery bacilli (Flexner type) were isolated from the stools of the cook and one of the kitchen helpers. This outbreak serves as an excellent example of the necessity of education of all food handlers as to the proper personal hygiene.

Hazardous Occupations

The State Factory Inspection Department for 1934-1935 showed that Hartford factories employed 24,653 persons exclusive of clerical help. This is 36 per cent of all persons gainfully occupied in that city. There are known to exist in the United States over 700 hazardous occupations and approximately 500 of these occur in Hartford. The protection of health of those engaged in industry against an injurious occupational environment is a responsibility of public health authorities. The expectancy of life of industrial employees has been shown to be about 7 years less than that of the average person.

Expansion of Hartford Health Board

Dr. Benjamin G. Horning, recently appointed Health Officer in Hartford, is planning a reorganization and expansion of the Health Department. Two new bureaus, Child Hygiene and Nursing, and the reorganization of the Bureau of Inspection, together with the addition of ten persons to the department's personnel, are proposed in the 1937-1938 budget estimates.

Plan Proposed for Medical Care of Automobile Accident Victims

Dr. Charles W. Comfort of New Haven has drafted a plan to be presented to the Financial Responsibility Commission. This plan provides for a State fund which would guarantee the payment of medical and hospital fees for the victims of automobile accidents when the motorist at fault is not financially responsible. It will require an additional registration fee for the automobile owner who carries no insurance and cannot show financial responsibility in other ways. The fund would also provide compensation to the victim for loss of time from work. The State, in turn, would have a claim against the motorist causing the accident and could refuse to grant him the right to drive again until he had reimbursed the State for payments made. The State might also collect from the motorist by a lien on any judgment returned against him. The Financial Responsibility Commission was expected to complete its report to the Governor in December. Organized medicine in Connecticut is opposed to compulsory liability insurance such as now exists in Massachusetts.

Optometrists Seek Eye Tests

The optometrists are said to be making a brave effort to secure annual eye examinations for every car driver before driving licenses are issued. They claim that nearly 50 per cent of driving accidents are due to visual defects. Stereopsis, or depth perception, and peripheral vision are of more importance to the driver than visual acuity.

Narcotics Destroyed

Three bushels of narcotic drugs, some of it taken from addicts who acquired it in illegitimate ways and more of it turned in to the State Department of Health as damaged or outdated stock of physicians and druggists, will be consigned officially to the furnace. Much of this housecleaning has been made possible under the new uniform narcotic act adopted by the General Assembly in 1935. Some packages of narcotics turned in bear Spanish-American War stamps. This stock represents in some cases uncalled for pharmacy products, narcotics damaged by the recent flood, and unwanted goods left behind when a pharmacist or physician has gone out of business or practice.

Marlborough Gets New Medical Examiner

Dr. Edwin Griswold of Glastonbury has been appointed Medical Examiner of Marlborough by Coroner Frank E. Healy. This appointment was made possible by the resignation of Dr. Lee J. Whittles from the Marlborough position. Dr. Whittles has been medical examiner for both Marlborough and Glastonbury for 13 years. He will continue to serve Glastonbury.

Dr. Pierson on Russia

Dr. Emily Pierson, health officer of Cromwell, is becoming a frequent lecturer on Soviet Russia. Dr. Pierson spent one year at the University of Moscow studying education and medicine and another year in travel throughout Russia observing various Soviet institutions. She presents moving pictures of schools, colleges, nurseries, churches, factories, col-

lective farms, playgrounds, camps and sanitaria. Dr. Pierson tells us that there is a sense of calm and security in Soviet Russia and she ascribes this to the medical care, free education (including college education with extra spending money), old age protection, and the assurance of a job provided by the State.

Hartford Shows Drop in Appendicitis Mortality

Hartford's appendicitis death rate dropped slightly during the past year and was lower than that of most other cities in the country, according to a national survey conducted by Dr. Frederick L. Hoffman. The city's record for 1935 was 10.7 per 100,000 population and in 1934 it was 10.9. The national average, based on studies in 180 cities, was 14.1 in 1935 as compared with 16.2 in 1934. The national figures are the lowest since 1918.

Infant Mortality

Of the four large cities in Connecticut, New Haven, Bridgeport, Waterbury and Hartford, the latter showed the highest average infant mortality rate for the years 1931 to 1935 inclusive. This situation is being used as one of the strongest arguments for the need of a Bureau of Child Hygiene within the City Board of Health in Hartford. The figures follow:

Bridgeport	43.9	per	1,000	population
Hartford	64.4	"	"	"
New Haven	44.1	"	"	"
Waterbury	53.2	"	"	"

Welfare Department Improvement Program

The Public Welfare Commission of Hartford has submitted to the Board of Finance a ten-year improvement program which includes three major undertakings: Reconstruction of the Isolation Hospital at a cost of $297,553; building of a nurses' home at $200,000; construction of an emergency lighting system for all institutions in the municipal hospital group at $500,000. The present Isolation Hospital, a wooden structure erected 20 years ago for a 5-year period, is a fire hazard, a financial white elephant, and entirely inadequate. Eighty per cent of the nurses in the hospital group are at present compelled to live outside because of lack of accommodations. An emergency lighting system is needed to insure heat in event of power failure. This was very apparent to all during the recent disastrous flood.

Vienna Physician Discusses Insulin Treatment in Dementia Praecox

Dr. M. Sakel of the University of Vienna, the discoverer of the so-called insulin shock treatment for dementia praecox, recently visited the Neuropsychiatric Institute of the Hartford Retreat. A conference was held on this form of treatment and there were present in addition to Connecticut's leading psychiatrists members of the faculties of Yale, Columbia, and Cornell Universities and the University of Munich.

Dr. Fritz Kant, associate professor of psychiatry at the University of Munich, has been appointed research associate at the Institute. He has been placed in charge of the evaluation and research involving investigation of insulin therapy in dementia praecox.

Head of St. Francis Hospital, Hartford, Dies

Mother Ann Valencia, head of St. Francis Hospital since its establishment in 1887, died in that institution November 29, 1936, at the age of 81. She was a most remarkable woman. Slight of body but indomitable of spirit, she was possessed of a keen mind that grappled with the problems of her institution. Mother Valencia entered the Order of the Sisters of St. Joseph at the age of 14 and was credited with having contributed the largest individual share to the growth and success of St. Francis Hospital. Until the very end her keen mind enabled her to issue orders to her subordinates as usual. Throngs found their way to the chapel where she lay in state to kneel at her bier. In the passing of this brilliant soul, the city and state have lost an untiring worker of extraordinary ability.

Sexton: Lewis A. Sexton, M.D., aged 60 years, who gained national reputation as a hospital administrator through his service as superintendent of the Hartford Hospital for nearly 20 years, died at his home at midnight December 2, 1936, after a long illness. In September Dr. Sexton was obliged, because of ill health, to relinquish his post as head of the hospital, and at a meeting on September 20 was named director emeritus. The post of superintendent was discontinued and Dr. Wilmar Mason Allen was appointed director of the hospital. As director emeritus Dr. Sexton continued to act in a consulting and advisory capacity.

From 1899 to 1901, Dr. Sexton was a student in the academic and pharmacologic departments of Vanderbilt University, Nashville, Tenn. He received his M.D. from Vanderbilt University in 1906. From 1901 to 1902 he served as assistant chemist of the Spurlock-Neal Company in Nashville.

He interned at Nashville City Hospital from 1906 to 1907 and from 1907 to 1908 at Riverside Hospital, New York City. From 1908 to 1914 Dr. Sexton served as resident physician at the Willard Parker Hospital, New York City.

Before coming to Hartford he served as assistant superintendent of Johns Hopkins Hospital, Baltimore, Md., for 3 years, from 1914 to 1917.

During the time of his administration of the Hartford Hospital, the number of beds at the hospital grew from 400 to 780 beds, until at present it is one of the largest private general hospitals in the country.

The Woman's Building, the Crane Building, the Hall-Wilson Laboratory for research, the Capewell X-Ray Building, the Barney Educational Building

for Nurses, the Cheney Memorial Library and the Ingalls Building were all added to the hospital while he was superintendent.

Extremely interested in the fireproofing of all hospitals, Dr. Sexton had contributed many articles on fire hazards in hospitals to medical journals.

He was instrumental in getting all insurance companies together to make a free survey of fire hazards in hospitals and through his efforts special policies and rates were drawn up. This move was made by insurance companies after an x-ray explosion in a Cleveland hospital caused a fire in the hospital and the death of several persons.

Dr. Sexton had the unusual honor of being named a member of the National Hospital Day Committee, consisting of 10 prominent physicians, to confer with former President Hoover on plans for National Hospital Day which is observed May 12.

At the time of his death, Dr. Sexton was president of the American Hospital Association, a position he had held for some time. He was former president of the New England Hospital Association, the Connecticut Hospital Association, and was a member of the American Medical Association, Connecticut Medical Society and Hartford Medical Society, and was an honorary member of the Western Hospital Association. His clubs included Farmington Country, Twentieth Century, Hartford Gun, Connecticut Historical Society, Sons of the American Revolution, East Glastonbury Fish and Game Association, Camp Fire Club of America, Phi Kappa Psi and Phi Chi. For some time he was a champion pistol shot.

Dr. Sexton was born March 25, 1876, in Tennessee, a son of the late William Robert and Mary (Sparkman) Sexton. He was married to Henrietta Stenz of New York City, April 19, 1916.

O'BRIEN: Thomas F. O'Brien, M.D., aged 52 years, former acting health officer of the city of Hartford and a member of the medical staff of St. Francis Hospital for many years, died at his home on November 30, 1936, after a long illness. He was born January 4, 1884, in Bridgeport, a son of Julia Agnes Ryan O'Brien and the late Timothy Peter O'Brien. After his elementary education, Dr. O'Brien had to struggle for his higher learning, finding it necessary to attend night school. At the age of 13, he went to work as cash boy in Reads's Department Store.

Seven years afterward, he entered St. Vincent's Hospital in Bridgeport, which had just opened, and became one of the first graduates of the hospital in 1907. In a short time, he entered the Mills Training School for male nurses at Bellevue Hospital, New York, for postgraduate work.

Lacking the prerequisites for entering a medical school, Dr. O'Brien practiced nursing in New York and Boston for some time and took courses paralleling high school courses. He was admitted to the College of Physicians and Surgeons in Baltimore, Md., and in 1916 obtained his medical degree there.

Through friendship with a fellow student, a Hartford man, Dr. O'Brien came to this city to intern at St. Francis Hospital. Completing a year's work at the hospital, he did considerable orthopedic work in New York and, at the outbreak of the World War, he was assistant superintendent of Undercliff Sanatorium in Meriden.

During the war, Dr. O'Brien was a lieutenant, junior grade, in the navy and served on the U.S. transport Neponset as ship's doctor. After the armistice he made ten trips on the transport Olivia, which was engaged in returning soldiers to this country.

Soon after his discharge from the navy, he became superintendent of Seaside Sanitarium, Niantic, where he remained until May, 1920.

In this period, Dr. O'Brien married Hilda Louise Stickney of Hartford whose father was for many years in charge of Trinity College Commons. Dr. and Mrs. O'Brien moved to Hartford, and from 1920 until 1934, he maintained a general practice here and served on the staff at St. Francis Hospital.

Dr. O'Brien entered the Health Department August 24, 1934, when he was appointed assistant superintendent and epidemiologist. When Superintendent C. P. Botsford retired in October, 1935, Dr. O'Brien became acting superintendent, a post he held until April this year. Dr. R. V. Boyce, then Health Board president, became acting superintendent and Dr. O'Brien was given an indefinite leave of absence because of ill health.

Dr. O'Brien was a member of Rau Locke Post, American Legion; Hartford Council and Bishop McMahon Assembly, Knights of Columbus; Bridgeport Lodge of Elks and the American Public Health Association.

He leaves his widow; his mother (Julia A. O'Brien Canny), of Bridgeport; two daughters, Mary Elizabeth and Hilda Frances O'Brien; two brothers, Joseph O'Brien and William Canny, of Bridgeport; two sisters, Mrs. R. W. Keeley, of Windsor, Ont., Canada, and Mrs. Raymond Maurer, of Point Barrow, Alaska, and several nieces and nephews.

HARTFORD HOSPITAL

At a dinner given recently in his honor as the newly appointed director of the Hartford Hospital by the managing directors and staff members, Dr. Wilmar M. Allen presented the three main problems that he considered the Hartford Hospital faces in the immediate future. First, how to provide medical care to all persons at a price that they can afford to pay; secondly, how to prevent the abuse of charity toward the medical profession and the hospital, and thirdly, how the Hartford Hospital can carry out these aims. In offering a solution for the first problem Dr. Allen suggested that it may be necessary to provide medical care on an insurance basis.

The Annual Meeting of the Hartford Hospital was held on November 12, 1936. Mr. Louis R. Cheney was again named president of the hospital. The staff elected included a few changes and additions, viz.: consulting pathologist and bacteriologist, Dr.

Wilmar M. Allen, anesthetist, Dr. Ralph M. Tovell; pathologist and bacteriologist, Dr. Ralph E. Kendall; clinical assistant physician, Dr. Lawrence P. Cogswell; clinical assistant ophthalmic surgeon, Dr. Arthur C. Unsworth; clinical assistant pediatrist, Dr. Frank P. Rogers; clinical assistant surgeon-at-large, Dr. Sidney S. Quarrier.

University of Healing Arts Closes

The University of Healing Arts, opened in Hartford a little more than a year ago as a school of drugless healing, suspended all instruction November 9, 1936, after State Health Commissioner Osborn had informed members of its faculty that they were violating a state law. Announcement of the suspension was made to the seventeen students the same day at the university's headquarters and announced publicly that evening by Dr. K. C. Hitchcock, secretary of the school. The decision followed receipt of legal advice from the school's counsel. It is understood that the school will attempt to put its affairs in order so that it may reopen.

Dr. Osborn called attention to violation of Section 2740 of the General Statutes of Connecticut which states that "no person shall give or attempt to give training in any branch of the healing arts or any subdivision thereof, or aid or participate in the same, without first having received permission to do so from the General Assembly".

The catalogue of the University announced courses in naturopathy, chiropractic, physical therapy and massage. All the students recently enrolled, of whom half are girls, are said to be studying naturopathy. The school is not recognized by either the chiropractic or the naturopathic examining boards. According to the catalogue, tuition fees are $200 a year in advance or $225 in installments, with an additional matriculation fee of $25 for students of naturopathy and chiropractic, a breakage deposit fee of $10 of which any balance would be refunded at the end of the school year, and variable laboratory fees "usually not exceeding" $25 a year.

Neuropsychiatric Institute of the Hartford Retreat

This hospital, the oldest of its kind in Connecticut and the third oldest in the United States, is entitled to exemption from taxation by the City of Hartford, according to a ruling recently made by the Assistant Corporation Counsel. The Retreat has always been exempt from taxation since its establishment 114 years ago but in June, 1936, the Assistant Corporation Counsel advised the assessors by letter that, in his opinion, the Retreat was not entitled to exemption. Now, after a more careful study of the facts, we find a reversal of opinion in the final conclusions reached by the Assistant Corporation Counsel.

The Mentally Deficient

Governor Cross has received a discouraging view of the burden of care for the mentally deficient in the report rendered by the Commission appointed to purchase the site for Connecticut's second hospital and training school for mental defectives. Experts estimate that two hospitals of the size of Mansfield, providing beds for 1,200 each, will not be sufficient to cover the need. A conservative estimate puts the number of mental defectives at 16,000 in Connecticut. Of these, 5,000 are said to require custodial care.

The danger of leaving such cases at large is often underestimated. New Haven has seen in recent years two murders perpetrated by youths of low mental capacity. The Jail Farm Commission in 1934 found that 33.2 per cent of the jail population was either feeble-minded or on the border line.

Hospitalization of Incurables

Connecticut is now having its attention brought to its lack of facilities for caring for its incurables. That such a group is just as deserving of public provision as are the insane, the feeble-minded and the tuberculous cannot be gainsaid. A number of states, notably Massachusetts and New York, have met the problem by providing State Hospitals for incurables. The situation is far more serious than the public in this State realizes.

Boucher: James J. Boucher, M.D., aged 61 years, secretary of the Hartford Board of Education, was killed in Columbia on November 14, 1936, by the discharge of a gun, accidentally fired as he fell in an attempt to scale a wall while hunting. Death was instantaneous. Dr. Boucher was born in East Windsor, received his primary education there, studied at Harvard University, and graduated from the College of Physicians and Surgeons in Baltimore, Maryland. He served his internship at St. Joseph's Hospital, Baltimore. He began practice in Hartford with his brothers and formerly was associated with one of them at the Wilson Sanatorium. Dr. Boucher was a member of St. Francis Hospital surgical staff for thirty years and was consulting surgeon to Mt. Sinai Hospital at the time of his death. He was a member of many medical organizations, of the American College of Surgeons, of the Hartford Lodge of Elks and of the Hartford Council, Knights of Columbus. Dr. Boucher leaves his wife, four daughters, and two brothers. Out of respect the elementary schools of Hartford were closed for one-half day and the high and junior high schools all day on November 17.

Thomas C. Hodgson, M.D., has been appointed health officer of Berlin to fill the unexpired term of Matthew H. Griswold, M.D.

Harold T. Oesau, M.D., has been appointed acting health officer of Stratford due to the death of Dr. Ruyter Howland, M.D.

Frank M. Dunn, M.D., has been appointed health officer of Waterford to fill the vacancy caused by the death of Ross E. Black, M.D.

ISOLATION UNIT FOR THE BURBANK HOSPITAL, FITCHBURG

An isolation building for the treatment of cases of diphtheria and scarlet fever has been built and equipped as an adjunct to the Burbank Hospital in Fitchburg. The plans were developed by James Purdon of Boston from ideas suggested by Dr. E. R. Lewis, former superintendent, with the assistance of Dr. Henry M. Pollock, superintendent of the Massachusetts Memorial Hospitals in Boston.

Every device for the treatment of cases of communicable diseases and the prevention of cross infections has been installed. The construction has been designed to avoid the likelihood of the retention of germs.

Fitchburg is to be congratulated in having an up-to-date plant for dealing with communicable diseases.

AN HONOR CONFERRED ON DR. L. M. S. MINER

The Alpha Omega dental fraternity has instituted the custom of an annual recognition of outstanding service to dentistry, and at a dinner at the Hotel Statler, Dr. Leroy Matthew Simpson Miner was presented with the first Achievement Award Medal by this organization.

Dr. Miner is the President of the American Dental Association and a Fellow of the Massachusetts Medical Society and the American Medical Association.

Dr. William Rich of New York, Chairman of the Award Committee, delivered the presentation address. Dr. Howard Marjerison, Dean of Tufts College Dental School, also spoke.

DR. SAMUEL H. EPSTEIN ADDRESSES THE SECTION OF PSYCHIATRY OF THE ROYAL SOCIETY OF MEDICINE

At a meeting of the Section of Psychiatry of the Royal Society of Medicine held in London on December 8, 1936, Dr. Samuel H. Epstein of Boston took part in the discussion of "The Follow-up Study of General Paralysis." The opening papers were read by Dr. F. L. Golla of The Maudesley Hospital and Dr. J. E. Nicole of Lancashire. Dr. Epstein is spending a year abroad on a grant from the Rockefeller Foundation.

CORRESPONDENCE

HORMONES AND HUMAN BEHAVIOR

December 17, 1936.

Editor, *New England Journal of Medicine*,

I was very much interested in the editorial on "Hormones and Human Behavior", published in your issue of December 10, 1936. The language used by Drs. Collip and Hoskins, on the presentation of the information corresponds very closely to the language used by the Hippocratic writers and by the late Dr. Galen on the proper crasis or mixture in the body of the four fluids, yellow bile, black bile, blood and phlegm. Of course these fluids are not hormones, but the ancient orders purely recognized that the condition of health was dependent upon the proper proportion and circulation of the fluids of the human body. Certainly history does repeat itself.

Yours very truly,

F. B. Lund, M.D.

319 Longwood Avenue,
Boston, Mass.

PERFORATION OF AN ULCER NEAR THE JUNCTION OF THE ESOPHAGUS AND STOMACH

December 1, 1936.

Editor, *New England Journal of Medicine*,

In the Cabot Case Records of the Massachusetts General Hospital, published in the *Journal* of May 2, 1935, there is a report of a case of perforation at the cardio-esophageal junction with continuing perforation through into the left pleural cavity. At about the same time we saw the case reported below and thought it unique. But without any thorough search of the literature we found a somewhat similar case in *Med. Klin.* (Aug. 12) 1932. In the *Brit. M. J.* (May 11) 1935 appeared the report of a case of perforation of the esophagus near the cardia and into the left pleural cavity.

F. P., 75 year old male, was admitted during the night to the Rhode Island Hospital.

C. C. Pain in the upper abdomen and left shoulder and arm for 3 hours. Admission note: Onset was sudden and of similar type, but more severe, than several "other attacks of indigestion". He is constipated by nature and has not had a good bowel movement for a week, but had a small movement yesterday. He was seen by Dr. Farrell at 11:00 p. m., with symptoms which have not increased in severity. B. P. 140/90. Temperature normal. Weak, barely audible heart sounds. The patient is conscious, rational and cooperative, and gives a fairly adequate history. There is moderate dyspnea on exertion. Nocturia 5 to 6 times.

P. H. He has been a high-strung nervous person all his life, but never required any medical attention until 3 years ago when he fell to the floor and had numbness and weakness of his left arm that returned to normal function in 2 or 3 weeks. There is no history of serious illnesses or operations. For the past 3 months or so the patient has had loss of appetite and has lost 15 pounds of weight in the past 6 months.

P. I. On the day of admission he vomited 4 times, once directly after a meal. In the evening he had pain in abdomen, shoulder and arm as noted above. Knows of no coffee-grounds vomitus, hematemesis, melena or blood-streaked stools.

P. E. Markedly emaciated old man in evident pain with respiratory rate of 30/35 and definite difficulty in expiring using all accessory muscles of respiration. Slight cyanosis of nails. Fairly quiet. Head not remarkable. Forcible venous pulsations in neck. Heart limits very difficult to demarkate. Sounds scarcely audible and rapid; evaluation difficult. Lungs

resonant. No definite rales. Expiration longer than inspiration. Abdomen silent. Tympany generalized. No dulness areas. No peristalsis seen. Upper abdomen rigid on both sides. Lower abdomen soft. Rectal: large non-tender prostate. Extremities not remarkable.

T. 101°. P. 130. R. 24.

Note by Dr. Cutts of the Cardiac Clinic. P. E. and EKG do not bear out diagnosis of cardiac trouble.

W. C. 20,000—88 per cent P. M. N.

X-ray examination of chest showed a large pneumothorax on the left and displacement of the heart and mediastinum to right, obscuring the right lung field but, so far as can be made out, the right chest appears normal. Plain films of the abdomen show no abnormality. Morph. gr. 1/6 given at 2:10 a. m. Saline and glucose intravenously ordered in forenoon and patient died while it was being given at 10:57 a. m.

At first we thought this a case of coronary sclerosis. By the time we had apparently eliminated this and established the fact of a left pneumothorax the patient was in extremis.

Pertinent extracts from autopsy report, restricted to chest:

The left lung is entirely collapsed and compressed against the mediastinum. In the pleural cavity of that side there are 1500 cc. of dark greyish rather foul-smelling fluid. There are a few fibrous adhesions at the apex of the left lung and these are made taut by the collapsed lung. At the medial surface of the inferior portion of the lower left lobe, the pleura (both the parietal and the visceral) is soft dark gray in color and it has a soft semigelatinous consistency. Elsewhere the pleural surfaces are glistening and pink. The right pleural cavity contains approximately 300 cc. of dark gray fluid and there are slight fibrous adhesions at the apex, but otherwise the pleural surfaces are pink and glistening. The right lung is collapsed as the thorax is open, but it is not compressed as is the left lung.

The mediastinum with its contents is displaced to the right from 3 to 4 cm.

The lining of the esophagus is grayish pink, glistening, and longitudinally folded. At the junction of the esophagus and cardiac end of the stomach the wall of the esophagus is somewhat thickened over an area approximately 8 mm. in width. Posterior to the esophagus and involving the loose connective tissue between the esophagus and aorta is a rather broad tract where the tissue is dark gray and of semigelatinous consistency. This tract extends from the region of the junction of the esophagus and the diaphragm posteriorly upward and slightly to the left to the region of the inferior portion of the hilus of the left lung. It is continuous with similar tissue which was described on the medial surface of the left lower lobe. The esophagus was cut flush with the diaphragm and the stomach was removed through an opening made through the diaphragm. In the posterior wall of the stomach in the region of the esophageal orifice is an ulcer, the upper end of which was cut across when the esophagus was removed. This ulcer is fusiform in outline, the long axis being parallel to the long axis of the esophagus. It is 1.2 cm. in length and .5 cm. in the transverse diameter and the edges are smooth and rounded. The base of the ulcer is approximately 3 mm. below the surface of the edges. There is a longitudinal fissure in the base which extends through the muscularis and into the surrounding connective tissue. The deepest portions are approximately 1 cm. below the surface and there the tissue is soft and dark gray in color. When the esophagus and the stomach are reconstructed this fissure in the base of the ulcer is seen to communicate with the esophagus. The pyloric orifice admits a finger with difficulty. Around the serosal surface there is evidence of constriction and there is increase of tough gray fibrous tissue. On the mucosal surface of the pylorus there are two ulcers, one approximately 8 mm. in diameter, the other approximately 8 mm. by 2 cm. There is a narrow partition between these ulcers and they practically surround the pyloric orifice. They are approximately 4 to 5 mm. in depth. Sections through the ulcers reveal increase in tough gray, fibrous-like tissue. The stomach contains a considerable amount of dark grayish brown fluid of similar apearance and odor to that found in the left pleural cavity.

Weiss and Mallory have described a clinical syndrome with vomiting followed by fissures or ulceration in the mucosa about the cardia and along the lower esophagus resulting in severe hemorrhages or perforation into the thoracic cavity. Evidently this occurs more frequently than has been previously recognized. The above case has been reported as it is thought that stressing this matter will put clinicians upon their guard and presumably early intervention may be of value in some of these cases.

PETER PINEO CHASE, M.D.
122 Waterman St.,
Providence, R. I.

RECENT DEATH

TILDEN—FRANK ELMER TILDEN, M.D., a retired physician of North Easton, died at his home, December 21, 1936, aged 83 years.

After graduating from the Harvard Medical School in 1876 he settled in North Easton, where he was born, and practiced there for 55 years. He was a Fellow of the Massachusetts Medical Society from 1876 to 1915. Dr. Tilden is survived by his widow, Mrs. Ellen Leonard Tilden.

NOTICES

MEDICAL CONFERENCE PROGRAM

Boston Dispensary, 25 Bennet Street, Boston—Lecture Hall, second floor, 9-10 a. m., January, 1937.

Tuesday, January 5—The Diagnosis and Treatment of Certain Foot Conditions—Dr. John D. Adams.

Wednesday, January 6—Hospital Case Presentation.—Dr. S. J. Thannhauser.

Thursday, January 7—Functional Nervous Disorders.—Dr. A. Warren Stearns.

Friday, January 8—Studies Pertaining to Joint Effusions.—Dr. Walter Bauer.

Saturday, January 9—Hospital Case Presentation.—Dr. S. J. Thannhauser.

Tuesday, January 12—Gastro-Intestinal Clinic.—Dr. K. S. Andrews.

Wednesday, January 13—Hospital Case Presentation.—Dr. S. J. Thannhauser.

Thursday, January 14—Urography and the Diagnosis of Diseases of the Upper Urinary Tract.—Dr. H. A. Chamberlin.

Friday, January 15—Carcinoma of the Colon and Rectum.—Dr. Frank H. Lahey.

Saturday, January 16—Hospital Case Presentation.—Dr. S. J. Thannhauser.

Tuesday, January 19—Treatment of Ambulatory Cases of Diabetes with Protamine Insulin.—Dr. Joseph Rosenthal.

Wednesday, January 20—Hospital Case Presentation.—Dr. S. J. Thannhauser.

Thursday, January 21—Transverse Rupture of the Aorta.—Dr. W. R. Crowe.

Friday, January 22—General Considerations of Coronary Thrombosis—Dr. Cadis Phipps.

Saturday, January 23—Hospital Case Presentation.—Dr. S. J. Thannhauser.

Tuesday, January 26—X-ray Demonstration.—Dr. Alice Ettinger.

Wednesday, January 27—Hospital Case Presentation.—Dr. S. J. Thannhauser.

Thursday, January 28—Social Service Case Presentation.—Miss E. R. Canterbury.

Friday, January 29—Recent Advances in the Surgery of the Chest.—Dr. Edward D. Churchill.

Saturday, January 30—Hospital Case Presentation.—Dr. S. J. Thannhauser.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, January 7, in the Amphitheater of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard Medical School and Physician-in-Chief, Peter Bent Brigham Hospital, will give a medical clinic. To it are cordially invited practitioners and medical students.

BOSTON'S COMMUNITY FUND CAMPAIGN

Boston physicians, as usual, are interested in the efforts of Greater Boston's Community Fund campaign in behalf of the 104 hospitals, health and social agencies, which will benefit in 1937.

Dr. Francis M. Rackemann is chairman of the physicians' group for the coming campaign and the vice-chairmen are as follows: Dr. Theodore L. Badger, Dr. Myles P. Baker, Dr. Edwin B. Dunphy, Dr. Thomas R. Goethals, Dr. Trygve Gundersen, Dr. E. Parker Hayden, Dr. Clark W. Heath, Dr. Richard B. King, Dr. James P. O'Hare, and Dr. Samuel N. Vose.

Robert Cutler, general chairman of the campaign, reports that 15,000 volunteers will help in collecting money and pledges in Boston proper and in the forty-two cities and towns of Metropolitan Boston which are served by the participating hospitals and agencies.

"The 1937 campaign," says Mr. Cutler, "is going to better last year's record, fine as it was. Last year more than 120,000 individuals pledged a total of three and three-quarters millions to the support of the agencies in the Community Federation of Boston. This year we are working for a greatly increased number of givers and an increased rate of giving.

"For a period of seven years now, many of our agencies have operated under deficits which have eaten into their capital funds or have been forced to curtail needed services. The successful oversubscription of last year's goal of $3,750,000 was the first step in establishing on a permanent basis, support for the needs of these agencies. The success, however, of the 1936 campaign has actually meant the payment in 1936 of only 77 per cent on the average, of these agencies' minimum operating requirements as established through the careful analysis of their submitted budgets by a representative citizens' Budget Committee. This year it is essential to oversubscribe a larger quota and thus to fill in this gap between the 77 per cent and the 100 per cent of minimum needs.

"We hope to enroll twice as many volunteer workers this year as in 1936. With an army of 15,000 volunteers, of whom more than 2,000 are already at work, we shall be able to rouse the whole community to the service of the agencies and to their needs. The results of campaigns in other cities point to a larger and finer support for the private charitable agencies in 1937 than ever before."

No class of prospective givers should be more interested in the success of this campaign than the doctors. Those with appointments on the staffs of hospitals financed in part by the campaign are absolutely dependent on its success for the continuance of the breadth of work and investigation now carried out in their hospitals. Those without such appointments depend on the same hospitals to care for their patients when financial or medical reasons make it impossible for the patient to get the best care as private patients. Doctors have always been generous of their time to the poor. If not for other reasons, purely to set an example as those receiving great indirect benefit, they must also give generously in money. If the doctors' quota should be set at double that of last year it could be reached without any man giving so generously as the employees of many large corporations do.

The organization is being formed now. The actual solicitation will take place at the end of January. Let every doctor do his part to show the community how we appreciate their work for us.

ANNOUNCEMENT

FRANK J. SULLIVAN, M.D., announces the opening of an office at 158 Mount Auburn Street, Cambridge, Mass.

REMOVALS

MAURICE G. EVANS, M.D., announces the removal of his office to 416 Marlborough Street, Boston, Mass.

NATHANIEL R. MASON, M.D., announces the removal of his office to 270 Commonwealth Avenue, Boston.

NOTICES OF MEETINGS

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The 96th Semi-Annual Meeting of the Essex North District Medical Society will be held Wednesday, January 6, at the First Universalist Church in Haverhill.

Dinner will be served at 12:30 p. m. followed by the business meeting. For the scientific program, Dr. Alexander P. Aitken will speak on the "Significance of Epiphyseal Injuries" and Dr. John Sproull will give "A Review of Some Features of Regional Ileitis."

BOSTON ORTHOPAEDIC CLUB

A meeting will be held in Ware Hall, Boston Medical Library, Monday, January 4, at 8:00 p. m.

1. Experiences in China; Orthopaedic and otherwise.—Dr. Phillips L. Greene.

2. Discussion and Further Observations—Dr. Van Gorder.

Dr. Greene is Dean of the Hunan Yale Medical College (Yale-in-China) and will speak not only on orthopaedic conditions, but also on subjects of general interest. His talk will be supplemented by Dr. Van Gorder, for many years Chief of the Orthopaedic Service at Peking Union Medical College.

An interesting and entertaining evening is assured, and wives and friends of members and guests are especially invited to attend.

The general medical profession is also invited.

SUMNER M. ROBERTS, M.D., *Secretary.*

WACHUSETT MEDICAL IMPROVEMENT SOCIETY

MEETING—WEDNESDAY, JANUARY 6, 1937

Bancroft Hotel, Worcester

6:30 p. m. Dinner.
7:15 p. m. Business Meeting.
7:30 p. m. Scientific Program.

Dr. Frank H. Lahey, head of Lahey Clinic, Boston, Mass.

Subject: "Hyperthyroidism — Hyperparathyroidism."

Physicians cordially invited to attend.

F. WASHBURN, M.D., *President,*
N. S. SCARCELLO, M.D., *Secretary-Treasurer.*

THE FAULKNER HOSPITAL CLINICAL MEETING

The next meeting will be held on January 7 at 5 p. m. In addition to the usual clinical-pathologic conference, Dr. William A. Rogers will speak on "Fractures of the Vertebral Column."

All physicians are invited.

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held at the Peter Bent Brigham Hospital, Monday, January 11, 1937, at 8:15 p. m.

PROGRAM

1. Three Clinical Case Records and Pathological Findings.—Dr. Samuel A. Levine.
2. Electrocardiographic Studies of Lead IV, using Nine Chest Positions.—Dr. A. W. Contratto.
3. Electrocardiographic Studies of Autopsied Cases of Congenital Heart Disease.—Dr. Maurice Schnitker.
4. Studies on Venous Pressure.—Dr. C. Sidney Burwell.
5. The Circulatory Adjustments in Arteriovenous Fistula.—Dr. J. A. Kennedy.

All members of the New England Heart Association and interested physicians are invited to attend.

JAMES M. FAULKNER, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, JANUARY 4, 1936

Monday, January 4—

*4 p. m. Physicians and medical students are cordially invited to attend a clinic presented by the Medical, Surgical and Orthopedic Services of the Infants' and Children's Hospitals in the Amphitheater of the Children's Hospital.

*8 p. m. Boston Orthopaedic Club. Boston Medical Library, 8 Fenway.

Tuesday, January 5—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. The Diagnosis and Treatment of Certain Foot Conditions. Dr. John D. Adams.

9:30 a. m. Massachusetts General Hospital. Thoracic Clinic—Ether Dome.

Wednesday, January 6—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

†12 m. Clinical-Pathologic Conference. Children's Hospital Amphitheater.

2 p. m. Massachusetts General Hospital. Psychiatric Clinic Outpatient Department.

4 p. m. - 5 p. m. Surgical Pathological Conference. Dr. Cutler and Dr. Wolbach, Peter Bent Brigham Hospital.

Thursday, January 7—

*8:30 - 9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and the Children's Hospitals, held this week at the Peter Bent Brigham Hospital.

9 a. m. Massachusetts General Hospital. Surgical Grand Rounds.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Functional Nervous Disorders. Dr. A. Warren Stearns.

9:15 a. m. Massachusetts General Hospital. Neurological Conference. Ether Dome.

12 m. Massachusetts General Hospital. Clinical-Pathologic Conference.

*3:30 p. m. Medical Clinic at the Peter Bent Brigham Hospital. Dr. Henry A. Christian.

5 p. m. Faulkner Hospital Clinical Meeting.

*6:30 p. m. Joint Meeting of the Boston Council of Social Agencies and the Boston Health League at the Hotel Vendome, Boston.

Friday, January 8—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Studies Pertaining to Joint Effusions. Dr. Walter Bauer.

10 a. m. Massachusetts General Hospital. Fracture Rounds.

12 m. Massachusetts General Hospital. Clinical Meeting of the Staff of the Children's Medical Service. Ether Dome.

Saturday, January 9—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

*10 a. m - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

Sunday, January 10—

4 p. m. Free public lecture at the Harvard Medical School, Building D, Longwood Avenue, Boston. "Air-Conditioning and Health," by Professor Philip Drinker.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

January 4—Boston Orthopaedic Club. See page 1321.

January 5-30—Boston Dispensary, Medical Conference Program. See page 1319.

January 6—Wachusett Medical Improvement Society. See page 1321.

January 7—Faulkner Hospital Clinical Meeting. See page 1321.

January 7—Medical Clinic at the Peter Bent Brigham Hospital. See page 1320.

January 7—Joint Meeting of the Boston Council of Social Agencies and the Boston Health League. See page 1259, issue of December 24.

January 10 - March 21—Sunday Afternoon Lectures at the Harvard Medical School. See page 1141, issue of December 10.

January 11—New England Heart Association. See page 1321.

January 14—Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

January 15—William Harvey Society, 8 p. m. in the Auditorium of the Beth Israel Hospital, Boston.

January 15—Boston Society for the Advancement of Gastroenterology. See page 1145, issue of December 10.

January 20—Illustrated Lecture at the Robert B. Brigham Hospital. See page 1256, issue of December 24.

February 3—American Social Hygiene Association. See page 1186, issue of December 17.

February 25, 26, 27—The New England Hospital Association. Hotel Statler, Boston.

March 30 - April 2—First International Conference on Fever Therapy. Postponement notice. See page 52, issue of July 2.

April 21-24—American Society for Experimental Pathology. See page 1075, issue of May 21.

October 25-29—American College of Surgeons. Chicago, Illinois.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

January 6—See page 1321.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

January 6—Meeting at the Danvers State Hospital, Hathorne, 5 p. m.

FRANKLIN DISTRICT MEDICAL SOCIETY

Will meet at the Weldon in Greenfield at 11 a. m. the second Tuesdays of January, March and May.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

January 13—Bear Hill Golf Club, Stoneham.
March 16—Danvers State Hospital, Danvers.
May 11—Bear Hill Golf Club, Stoneham.

KENNETH L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

January 19—8:15 p. m. The Peter Bent Brigham Hospital. Communications and Case Presentations by the Staff. Suggested title—"Abdominal Pain from the Medical and Surgical Standpoint." Details of program to be announced.

February 23—Time, place and details of program to be announced.

March 30—8:15 p. m. New England Deaconess Hospital. A Symposium on Diabetes entitled "A Survey of the Diabetic Work of the George F. Baker Clinic in the New England Deaconess Hospital." Communications and Case Presentations by the Staff. Drs. Elliott P. Joslin, Howard F. Root, Priscilla White, Alexander Marble and Allen P. Joslin.

May—Annual Meeting. Details to be announced.

Note: The Censors will meet for the examination of candidates on the first Thursday of May, 1937. Fee of $10.00 is payable at the time of examination. Application blanks may be obtained by writing the Secretary, furnishing name, address and name of school of graduation in medicine. Application must be made at least three weeks prior to date of examination. Candidates whose applications are on file will receive proper notices.

FRANK S. CRUICKSHANK, M.D., Secretary.
1247 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

January 21—11 a. m. Bridgewater State Farm.
March 18—11 a. m. Brockton Hospital.
April 15—Annual Meeting. 11 a. m. Ducy Hospital.
May 20—11 a. m. Lakeville State Sanatorium.

FRED F. WEINER, M.D., Secretary.
231 Main Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

January 27—Boston Medical Library. 8:15 p. m. Joint Meeting with the Boston Medical Library. "Anthropology." Dr. Carleton S. Coon.

March 31—Boston Medical Library. 8:15 p. m. "Social Insurance—It Affects the Medical Profession." Dr. Charles E. Mongan. Discussion: Dr. Channing Frothingham.

April 28—Annual Meeting. Boston Medical Library. 8:15 p. m. "Problems in Surgical Diagnosis." Dr. Howard M. Clute.

CONRAD WESSELHOEFT, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

January 13—Worcester City Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

February 10—Worcester State Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

March 10—The Memorial Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

April 14—Worcester Hahnemann Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

May 6—At 4:30 in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the spring meeting of the Board of Censors.

Wednesday Afternoon and Evening, May 12—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

January 27—Meeting at the Leominster Hospital, 4:30 p. m.

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